L9 ANSWER 1 OF 29 PCTFULL COPYRIGHT 2003 Univentio
ACCESSION NUMBER: 1998031709 PCTFULL ED 20020514
TITLE (ENGLISH): **ANTIBODIES** THAT BIND TO THE **NIDOGEN** -BINDING DOMAIN OF LAMININ, THEIR PRODUCTION AND USE
TITLE (FRENCH): ANTICORPS QUI SE LIENT AUX DOMAINES DE LIAISON DE NIDOGENE DE LA LAMININE, LEUR PRODUCTION ET LEUR UTILISATION
INVENTOR(S): GERL, Martin
PATENT ASSIGNEE(S): HOECHST AKTIENGESELLSCHAFT; GERL, Martin
LANGUAGE OF PUBL.: German
DOCUMENT TYPE: Patent
PATENT INFORMATION:

NUMBER	KIND	DATE
WO 9831709	A1	19980723

DESIGNATED STATES
W: AU BR CA CN CZ HU ID IL JP KR MX PL RU TR US AT BE CH DE DK ES FI FR GB GR IE IT LU MC NL PT SE
APPLICATION INFO.: WO 1997-EP7241 A 19971222
PRIORITY INFO.: DE 1997-197 01 607.3 19970117

ABEN Monoclonal and polyclonal antibodies are disclosed as well as parts thereof which bind specifically to the nidogen-binding domain of laminin, as well as a process for producing the same and their use as medicaments, as diagnostic agents for detecting laminin isoforms and as model substances for developing and evaluating substances that influence the **nidogen**-laminin interaction. The disclosed **antibodies** or their parts bind preferably to the 'gamma'1 III 4-domain of laminin, in particular in the highly preserved area of loops a and c, and can inhibit the association of laminin and nidogen.

ABFR L'invention concerne des anticorps monoclonaux et polyclonaux et leurs parties qui se lient specifiquement au domaine de liaison de nidogene de la laminine, leur procede de production et leur utilisation comme medicaments, comme agents de diagnostic permettant de detecter des isoformes de la laminine et comme substances modeles permettant de developper et d'evaluer des substances qui affectent l'interaction entre le nidogene et la laminine. Ces anticorps ou leurs parties se lient de preference au domaine 'gamma'1 III 4 de la laminine, surtout dans le domaine tres conserve des boucles a et c, et peuvent inhiber l'association de la laminine au nidogene.

L9 ANSWER 2 OF 29 CANCERLIT DUPLICATE 1
ACCESSION NUMBER: 1998311650 CANCERLIT
DOCUMENT NUMBER: 98311650 PubMed ID: 9647658
TITLE: The laminin-nidogen complex is a ligand for a specific splice isoform of the transmembrane protein tyrosine phosphatase LAR.
AUTHOR: O'Grady P; Thai T C; Saito H
CORPORATE SOURCE: Dana-Farber Cancer Institute and Department of Biological Chemistry and Molecular Pharmacology, Harvard Medical School, Boston, Massachusetts 02115, USA.
CONTRACT NUMBER: GM53415 (NIGMS)
SOURCE: JOURNAL OF CELL BIOLOGY, (1998 Jun 29) 141 (7) 1675-84. Journal code: 0375356. ISSN: 0021-9525.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English
FILE SEGMENT: MEDLINE; Priority Journals
OTHER SOURCE: MEDLINE 1998311650
ENTRY MONTH: 199808
ENTRY DATE: Entered STN: 19980910
Last Updated on STN: 19980910

AB Leukocyte antigen-related protein (LAR) is a prototype for a family of transmembrane protein tyrosine phosphatases whose extracellular domain is composed of three Ig and several fibronectin type III (FnIII) domains. Complex alternative splicing of the LAR-FnIII domains 4-8 has been observed. The extracellular matrix laminin-nidogen complex was identified as a ligand for the LAR-FnIII domain 5 (Fn5) using a series of GST-LAR-FnIII domain fusion proteins and testing them in in vitro ligand-binding assays. LAR- laminin-nidogen binding was regulated by alternative splicing of a small exon within the LAR-Fn5 so that inclusion of this exon sequence resulted in disruption of the laminin-nidogen-binding activity. Long cellular processes were observed when HeLa cells were plated on laminin-**nidogen**, but not when plated on a fibronectin surface. Indirect immunofluorescent **antibody** staining revealed high expression of LAR in a punctate pattern, throughout the length of these cellular processes observed on laminin-**nidogen** . **Antibody**-induced cross-linking of LAR inhibited formation of these cellular processes, and inhibition was correlated with changes in cellular actin cytoskeletal structure. Thus, LAR-laminin-nidogen binding may play a role in regulating cell signaling induced by laminin-nidogen, resulting in cell morphological changes.

L9 ANSWER 3 OF 29 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 1998406162 MEDLINE
DOCUMENT NUMBER: 98406162 PubMed ID: 9733643
TITLE: Nidogen-2: a new basement membrane protein with diverse binding properties.
AUTHOR: Kohfeldt E; Sasaki T; Gohring W; Timpl R
CORPORATE SOURCE: Max-Planck-Institut fur Biochemie, D-82152 Martinsried, Germany.
SOURCE: JOURNAL OF MOLECULAR BIOLOGY, (1998 Sep 11) 282 (1) 99-109. Journal code: 2985088R. ISSN: 0022-2836.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-AJ223500
ENTRY MONTH: 199810
ENTRY DATE: Entered STN: 19981021
Last Updated on STN: 19981021
Entered Medline: 19981015

AB Human nidogen-2 was cloned and sequenced (1375 residues) and found to share 46% sequence identity and a similar domain arrangement with the previously characterized basement membrane protein nidogen-1. Recombinant nidogen-2 was purified as a 200 kDa protein from transfected mammalian cell medium, showed a high level of N and O-glycosylation, and could be clearly distinguished from **nidogen**-1 (150 kDa) by specific **antibodies**. Electron microscopy demonstrated that the two isoforms have a similar shape, consisting of three globular domains connected by two threads, but differ somewhat in length. Northern blots and immunological assays demonstrated co-expression of the nidogens in various tissues and cultured cells. Immunofluoresence revealed colocalization in vessel walls and other basement membrane zones but some differences in heart and skeletal muscle. Nidogen-2 interacted with collagens I and IV, and perlecan at a comparable level to nidogen-1 but failed to bind to fibulins. Nidogen-2 bound to laminin-1, but only moderately to the epitope on the laminin gamma1 chain, which promotes high-affinity binding of nidogen-1. Both nidogens were cell-adhesive for a restricted number of cell lines, with nidogen-2 having a higher

activity. Together, these data suggest that nidogen-2 can compensate for some but not all functional activities ascribed to nidogen-1.

L9 ANSWER 4 OF 29 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 97195710 MEDLINE
DOCUMENT NUMBER: 97195710 PubMed ID: 9043083
TITLE: Importance of nidogen binding to laminin gamma1 for branching epithelial morphogenesis of the submandibular gland.
AUTHOR: Kadoya Y; Salmivirta K; Talts J F; Kadoya K; Mayer U; Timpl R; Ekblom P
CORPORATE SOURCE: Department of Animal Physiology, Uppsala University, Biomedical Center,Sweden.
SOURCE: DEVELOPMENT, (1997 Feb) 124 (3) 683-91.
Journal code: 8701744. ISSN: 0950-1991.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199703
ENTRY DATE: Entered STN: 19970407
Last Updated on STN: 20000303
Entered Medline: 19970325

AB Epithelial-mesenchymal interactions are major driving forces for the development of most solid organs. The importance of these interactions was first shown for the embryonic submandibular gland more than 40 years ago. We here present evidence that interactions between two basement membrane components, nidogen (entactin) and laminin gamma1 chain, could be important for epithelial-mesenchymal interactions in this gland. Nidogen mRNA was detected by in situ hybridization in the mesenchyme, and yet the protein was detected in epithelial and endothelial basement membranes. The role of **nidogen**-laminin interactions for epithelial morphogenesis was studied by applying **antibodies** to submandibular gland organ cultures. **Antibodies** reacting strongly with the **nidogen**-binding site of laminin gamma1 chain drastically perturbed branching epithelial morphogenesis. Electron microscopy of the epithelial-mesenchymal interface showed that blocking antibodies disrupted the formation of the basement membrane. Epidermal growth factor was shown to increase the expression of **nidogen** in mesenchyme, and could counteract the effect of the blocking **antibodies**. We suggest that nidogen could be an important mesenchymal factor for submandibular gland development.

L9 ANSWER 5 OF 29 USPATFULL
ACCESSION NUMBER: 96:14906 USPATFULL
TITLE: Two non-contiguous regions contribute to nidogen binding to a single EGF-like motif of the laminin .gamma.1 chain
INVENTOR(S): Fox, Jay W., Charlottesville, VA, United States
Timpl, Rupert, Martinsried, Germany, Federal Republic of
PATENT ASSIGNEE(S): The University Of Virginia Patent Foundation, Charlottesville, VA, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 5493008		19960220	
APPLICATION INFO.:	US 1994-288728		19940815	(8)

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Warden, Jill
ASSISTANT EXAMINER: Prickril, Benet
LEGAL REPRESENTATIVE: Oblon, Spivak, McClelland, Maier & Neustadt

NUMBER OF CLAIMS: 12
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 3 Drawing Figure(s); 2 Drawing Page(s)
LINE COUNT: 981
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB High affinity binding of nidogen to laminin is mediated by an EGF-like repeat .gamma.1III4 of the mouse laminin .gamma.1 chain and has now been restricted to two short non-contiguous regions of its 56 residue sequence by use of synthetic peptides and recombinant mutants. Disulfide loop a,b of the repeat and a modified loop a,c could completely inhibit binding, with a 5,000-fold or 300-fold reduced affinity, respectively. Synthetic loops c and d lacked inhibitory activity. Some binding contribution of Try819 in loop c was, however, shown by mutation and side chain modification. Together with studies of loop chimeras, this indicated a distinct cooperativity between the two binding sites. The major binding site of loop a was localized to the heptapeptide NIDPNAV (position 798-804). A change of Asp800 to Asn or Ala803 to Val caused a strong reduction in binding activity, while only small effects were observed for the changes Pro801 to Gln and Ile799 to Val. The latter replacement corresponds to the single substitution found in the same region of the Drosophila laminin .gamma.1 chain. However, the changes Asn802 to Ser or Val804 to Ser, both known to exist in the laminin .gamma.2 chain, were deleterious mutations. This demonstrated conservation of binding structure in laminins of distantly related species, but not between homologous chains of laminin isoforms.

L9 ANSWER 6 OF 29 PCTFULL COPYRIGHT 2003 Univentio
ACCESSION NUMBER: 1996004926 PCTFULL ED 20020514
TITLE (ENGLISH): TWO NON-CONTIGUOUS REGIONS CONTRIBUTE TO NIDOGEN BINDING TO A SINGLE EGF-LIKE MOTIF OF THE LAMININ 'gamma'1 CHAIN
TITLE (FRENCH): DEUX REGIONS NON CONTIGUES CONTRIBUANT A LA LIAISON NIDOGENE AVEC UN MOTIF UNIQUE DU TYPE EGF DE LA CHAINE 'gamma'1 DE LA LAMININE
INVENTOR(S): FOX, Jay, W.;
TIMPL, Rupert
PATENT ASSIGNEE(S): THE UNIVERSITY OF VIRGINIA PATENT FOUNDATION
LANGUAGE OF PUBL.: English
DOCUMENT TYPE: Patent
PATENT INFORMATION:

NUMBER	KIND	DATE
WO 9604926	A1	19960222

DESIGNATED STATES
W: AU CA JP AT BE CH DE DK ES FR GB GR IE IT LU MC NL PT SE
APPLICATION INFO.: WO 1995-US9693 A 19950811
PRIORITY INFO.: US 1994-288,728 19940815

ABEN The present invention relates to peptide antagonists which specifically prevent laminin
interaction with nidogen. Laminin is a major cell-adhesive and structural protein of basement
membranes and other extracellular structures occurring as various isoforms of 600-900 kDa, and
contains a single high affinity binding site for the 150 kDa basement membrane protein nidogen. The
peptide antagonists of this invention may be applied to in vitro studies of organ development or as
therapeutic agents for clinical use.
ABFR Cette invention concerne des antagonistes de peptides qui empechent de maniere specifique
l'interaction de la laminine avec le nidogene. La laminine est une proteine majeure de structure et
d'adhesion cellulaire des membranes basales et d'autres structures

extracellulaires se presentant
sous diverses isoformes de 600-900 kDa, et contient un seul et unique site de liaison a forte
affinite pour le nidogene de proteine de membrane basale a 150 kDa. On peut utiliser les
antagonistes de peptides de cette invention dans le cadre des recherches in vitro sur la croissance
d'organe ou comme agents therapeutiques destines a un usage clinique.

L9 ANSWER 7 OF 29 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 96007609 MEDLINE
DOCUMENT NUMBER: 96007609 PubMed ID: 7561165
TITLE: Skin fibroblasts are the only source of nidogen during early basal lamina formation in vitro.
AUTHOR: Fleischmajer R; Schechter A; Bruns M; Perlish J S; Macdonald E D; Pan T C; Timpl R; Chu M L
CORPORATE SOURCE: Department of Dermatology, Mount Sinai School of Medicine, New York, New York 10029, USA.
SOURCE: JOURNAL OF INVESTIGATIVE DERMATOLOGY, (1995 Oct) 105 (4) 597-601.
Journal code: 0426720. ISSN: 0022-202X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199511
ENTRY DATE: Entered STN: 19951227
Last Updated on STN: 20000303
Entered Medline: 19951114

AB The purpose of this study was to determine whether nidogen, the linkage protein of the basal lamina, is of epidermal or dermal origin. The development of the basal lamina was studied in an in vitro skin model. Preputial fibroblasts seeded onto a nylon mesh attached, proliferated, and developed a rich extracellular matrix (dermal model). Preputial keratinocytes were added to the dermal model to form a keratinocyte dermal model that ultrastructurally resembled in many respects human skin. Ultrastructural analysis revealed early stages of dermal development, including an incomplete basal lamina, aggregates of dermal filamentous material connecting to the lamina densa, bundles of 10-nm microfibrils, formation of premature hemidesmosomes, anchoring filaments, and anchoring fibrils. The cell origin of nidogen was determined in the dermal model and in the epidermal and dermal components of the keratinocyte dermal model. Specific **antibodies** and a cDNA probe for **nidogen** were used for immunofluorescence microscopy, Western and Northern blots, and for in situ hybridization studies. Our data show that fibroblasts are the only source of nidogen during early basal lamina formation. Although fibroblasts can synthesize nidogen and deposit it in the dermal matrix, no basal lamina will form unless they are recombined with keratinocytes. This suggests that the epidermis plays a major regulatory role in the production and assembly of nidogen into the basal lamina.

L9 ANSWER 8 OF 29 MEDLINE DUPLICATE 5
ACCESSION NUMBER: 95009530 MEDLINE
DOCUMENT NUMBER: 95009530 PubMed ID: 7925005
TITLE: Role of mesenchymal nidogen for epithelial morphogenesis in vitro.
AUTHOR: Ekblom P; Ekblom M; Fecker L; Klein G; Zhang H Y; Kadoya Y; Chu M L; Mayer U; Timpl R
CORPORATE SOURCE: Department of Animal Physiology, Uppsala University, Sweden.
CONTRACT NUMBER: AR 38923 (NIAMS)
SOURCE: DEVELOPMENT, (1994 Jul) 120 (7) 2003-14.
Journal code: 8701744. ISSN: 0950-1991.
PUB. COUNTRY: ENGLAND: United Kingdom

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199411
ENTRY DATE: Entered STN: 19941222
Last Updated on STN: 19941222
Entered Medline: 19941101

AB Recent biochemical studies suggested that the extracellular matrix protein nidogen is a binding molecule linking together basement membrane components. We studied its expression and role during development. By immunofluorescence and northern blotting, nidogen was found early during epithelial cell development of kidney and lung. Yet, in situ hybridization revealed that nidogen was not produced by epithelium but by the adjacent mesenchyme in both organs. Binding of mesenchymal nidogen to epithelial laminin may thus be a key event during epithelial development. This is supported by antibody perturbation experiments. **Antibodies** against the **nidogen** binding site on laminin B2 chain perturbed epithelial development in vitro in embryonic kidney and lung. Mesenchymal nidogen could be important for early stages of epithelial morphogenesis.

L9 ANSWER 9 OF 29 MEDLINE DUPLICATE 6
ACCESSION NUMBER: 95051016 MEDLINE
DOCUMENT NUMBER: 95051016 PubMed ID: 7962110
TITLE: Influence of nidogen complexed or not with laminin on attachment, spreading, and albumin and laminin B2 mRNA levels of rat hepatocytes.
AUTHOR: Levavasseur F; Mayer U; Guillouzo A; Clement B
CORPORATE SOURCE: Unite de Recherches Hepatologiques, INSERM U-49, Hopital Pontchaillou, Rennes, France.
SOURCE: JOURNAL OF CELLULAR PHYSIOLOGY, (1994 Nov) 161 (2) 257-66. Journal code: 0050222. ISSN: 0021-9541.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199412
ENTRY DATE: Entered STN: 19950110
Last Updated on STN: 19950110
Entered Medline: 19941228

AB Nidogen/entactin is a Mr = 150,000 glycoprotein which is present within basement membranes in a noncovalent stable complex with laminin. We have studied the effects of nidogen/entactin complexed or not with laminin on attachment, spreading, and functions of adult rat hepatocytes in primary culture. Freshly isolated hepatocytes attached on either recombinant or EHS-derived nidogen, although to a lesser extent than on laminin/nidogen complex, laminin, and E8 and P1 fragments of laminin. Hepatocytes bound on a nidogen fragment bearing the N-terminal and rod-like domains but not on either the N-terminal globules or the rod-like domain which contains a RGD sequence. Attachment of hepatocytes on nidogen and laminin/**nidogen** complex was inhibited by anti-beta 1 integrin **antibodies**. Hepatocytes remained rounded on nidogen and laminin, whereas they rapidly spread on laminin/nidogen complex and collagen IV. Nidogen, laminin, and laminin/nidogen complex transiently maintained high steady-state albumin mRNA levels in cultured hepatocytes, but a decrease in albumin mRNA content was observed after 24 h, independently of the substrates. Actinomycin D and cycloheximide treatment indicated that the transient effect of these substrates on albumin expression was related to post-transcriptional mechanisms. Laminin B2 mRNAs were not detectable in freshly isolated hepatocytes but were expressed in 4 h hepatocyte cultures. After 24 h, a dramatic increase in the steady-state level of laminin B2 mRNA was found in hepatocytes cultured on nidogen and laminin/nidogen complex. This effect was slightly prevented in hepatocytes plated on laminin. These results show that interactions of

hepatocytes with nidogen/entactin in vitro result only in a transient modulation of hepatocyte functions.

L9 ANSWER 10 OF 29 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.DUPLICATE 7

ACCESSION NUMBER: 1993:342697 BIOSIS
DOCUMENT NUMBER: PREV199396039697
TITLE: A single EGF-like motif of laminin is responsible for high affinity nidogen binding.
AUTHOR(S): Mayer, Ulrike; Nischt, Roswitha; Poeschl, Ernst; Mann, Karlheinz; Fukuda, Katsunori; Gerl, Martin; Yamada, Yoshihiko; Timpl, Rupert (1)
CORPORATE SOURCE: (1) Max-Planck-Inst. Biochem., D-8033 Martinsried Germany
SOURCE: EMBO (European Molecular Biology Organization) Journal, (1993) Vol. 12, No. 5, pp. 1879-1885. ISSN: 0261-4189.
DOCUMENT TYPE: Article
LANGUAGE: English

AB A major nidogen binding site of mouse laminin was previously localized to about three EGF-like repeats (Nos 3-5) of its B2 chain domain III (M.Gerl et al. (1991) Eur. J. Biochem., 202, 167). The corresponding cDNA was amplified by polymerase chain reaction and inserted into a eukaryotic expression vector tagged with a signal peptide. Stably transfected human kidney cell clones were shown to process and secrete the resulting fragment B2II3-5 in substantial quantities. It possessed high binding activity for recombinant nidogen in ligand assays, with an affinity comparable with that of authentic laminin fragments. In addition, complexes of B2III3-5 and nidogen could be effectively converted into a covalent complex by cross-linking reagents. Proteolytic degradation of the covalent complex demonstrated the association of BIII3-5 with a apprx 80 residue segment of nidogen domain G3 to which laminin binding has previously been attributed. The correct formation of most of the 12 disulfide bridges in B2III3-5 was indicated from its protease resistance and the complete loss of cross-reacting epitopes as well as of nidogen-binding activity after reduction and alkylation. Smaller fragments were prepared by the same recombinant procedure and showed that combinations of EGF-like repeats 3-4 and 4-5 and the single repeat 4 but not repeats 3 or 5 possess full nidogen-binding activity. This identifies repeat 4 as the only binding structure. The sequence of repeat 4 is well conserved in the human and in part in the Drosophila laminin B2 chain. It is further shown that **antibodies** against B2III3-5 inhibit laminin binding to **nidogen**, indicating that repeat 4 represents the only high affinity binding site of laminin.

L9 ANSWER 11 OF 29 MEDLINE DUPLICATE 8

ACCESSION NUMBER: 93146648 MEDLINE
DOCUMENT NUMBER: 93146648 PubMed ID: 8425764
TITLE: Myoepithelial and basement membrane antigens in benign and malignant human breast tumors.
AUTHOR: Guelstein V I; Tchypysheva T A; Ermilova V D; Ljubimov A V
CORPORATE SOURCE: Cancer Research Center, Russian Academy of Medical Sciences, Moscow.
SOURCE: INTERNATIONAL JOURNAL OF CANCER, (1993 Jan 21) 53 (2) 269-77. Journal code: 0042124. ISSN: 0020-7136.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199303
ENTRY DATE: Entered STN: 19930312
Last Updated on STN: 19930312
Entered Medline: 19930304

AB Serial cryostat sections of 160 human breast lesions and of 9 lymph-node

metastases were studied by indirect immunofluorescence. We used monoclonal antibodies (MAbs) to lining-epithelium-specific keratin 8 and to myoepithelium-specific keratin 17 in combination with polyclonal and monoclonal **antibodies** to major basement membrane components, laminin, collagen type IV, entactin/**nidogen**, and large heparan sulfate proteoglycan (perlecan) core protein. Continuous basement membranes adjacent to a basal layer of keratin-17-positive myoepithelial cells were typical for normal, benign and in situ carcinomatous structures. In invasive and metastatic structures, always formed by keratin-8-positive tumor cells, basement membranes were found only rarely and with conspicuous fragmentations. This lack of basement membranes correlated with loss of myoepithelium identified by staining for keratin 17. In comedo structures of invasive ductal carcinomas and in papillary carcinomas, fibrovascular complexes with numerous blood vessels and deposition of basement membrane material were often seen in the stroma. Immunomorphological analysis of 41 cases of doubtful diagnosis at intra-operative biopsy was also performed. A combination of MAbs to keratins 8 and 17, and to basement membrane components, made it possible to distinguish between morphologically similar benign and malignant proliferations and to detect single-cell invasion of the stroma. This combination of antibodies may be recommended as an auxiliary immunomorphological tool for differential diagnosis of intra-operative breast biopsies in dubious cases.

L9 ANSWER 12 OF 29 MEDLINE DUPLICATE 9
ACCESSION NUMBER: 93238676 MEDLINE
DOCUMENT NUMBER: 93238676 PubMed ID: 8477687
TITLE: Ascidian entactin/nidogen. Implication of evolution by shuffling two kinds of cysteine-rich motifs.
AUTHOR: Nakae H; Sugano M; Ishimori Y; Endo T; Obinata T
CORPORATE SOURCE: Advanced Research Laboratory, Research and Development Center, Toshiba Corporation, Japan.
SOURCE: EUROPEAN JOURNAL OF BIOCHEMISTRY, (1993 Apr 1) 213 (1) 11-9.
Journal code: 0107600. ISSN: 0014-2956.
PUB. COUNTRY: GERMANY: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-D14038; GENBANK-L09679; GENBANK-L09680; GENBANK-L09681; GENBANK-L09682; GENBANK-L09683; GENBANK-X57950; GENBANK-X70793; GENBANK-X70999; GENBANK-X71000
ENTRY MONTH: 199305
ENTRY DATE: Entered STN: 19930611
Last Updated on STN: 20000303
Entered Medline: 19930521

AB Entactin/nidogen, a major component of the basement membrane, has a domain structure comprising three globular domains, and thread-like and rod-like domains connecting them. It contains six epidermal-growth-factor-(EGF)-like motifs and one thyroglobulin-like motif. In the present study, ascidian entactin/**nidogen** has been identified by a monoclonal **antibody** technique. We prepared anti-(ascidian entactin/nidogen) IgG, named anti-AsEnt1, then cloned the cDNA of ascidian entactin/nidogen using anti-AsEnt1 as a probe, and determined its entire sequence. Mainly because the deduced amino acid sequence exhibited high similarity to mouse entactin and human nidogen, and because the antigen localized in basement membrane of ascidian body-wall muscle, we have concluded that the antigen anti-AsEnt1 corresponds to the ascidian entactin/nidogen homologue. The deduced amino acid sequence of ascidian entactin/nidogen clearly showed that the ascidian homologue also has a domain structure. However, the ascidian homologue lacked the thread-like domain, and the rod-like domain differed from that of mouse entactin in composition, consisting of two kinds of cysteine-rich motifs, that is, the

EGF-like motif and the thyroglobulin-like motif. These results suggest that entactin/nidogen have evolved by modifying the domains, especially by shuffling the two kinds of cysteine-rich motifs.

L9 ANSWER 13 OF 29 MEDLINE DUPLICATE 10
ACCESSION NUMBER: 92165419 MEDLINE
DOCUMENT NUMBER: 92165419 PubMed ID: 1371500
TITLE: Distribution of individual components of basement membrane in human colon polyps and adenocarcinomas as revealed by monoclonal antibodies.
AUTHOR: Ljubimov A V; Bartek J; Couchman J R; Kapuller L L; Veselov V V; Kovarik J; Perevoshchikov A G; Krutovskikh V A
CORPORATE SOURCE: All-Union Cancer Research Center, USSR AMS, Moscow.
CONTRACT NUMBER: AR36457 (NIAMS)
SOURCE: INTERNATIONAL JOURNAL OF CANCER, (1992 Feb 20) 50 (4) 562-6.
Journal code: 0042124. ISSN: 0020-7136.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199203
ENTRY DATE: Entered STN: 19920417
Last Updated on STN: 19980206
Entered Medline: 19920330

AB Double-label immunofluorescence was used to monitor basement-membrane composition and integrity in 22 human colon polyps, 36 adenocarcinomas and 2 metastases. Cryostat sections were stained with polyclonal anti-laminin anti-serum combined with monoclonal **antibodies** (MAbs) to all major basement-membrane components (laminin, entactin/**nidogen**, collagen type IV and large heparan sulfate proteoglycan), as well as to keratin 8. In all adenocarcinomas, including mucinous, basement membranes were altered more at the invasive front than in the parenchyma. The degree of this alteration was inversely correlated with the level of tumor differentiation. An uncoordinated loss of basement membrane components (dissociation of markers), previously described by us in rat colon adenocarcinomas, was also found in human tumors. In the great majority of adenocarcinomas a pronounced stromal reaction was seen. It was manifested by the presence of fibrillar deposits of basement-membrane components, mainly of collagen type IV and/or heparan sulfate proteoglycan. This reaction was never observed in polyps and may be derived from myofibroblasts reported to accumulate in colon cancer stroma. The combined use of antibodies to basement-membrane components and to a specific keratin may constitute an adequate immunohistochemical test for the presence of invasion, and may be useful in the histologic analysis of polyps, especially in dubious cases.

L9 ANSWER 14 OF 29 MEDLINE DUPLICATE 11
ACCESSION NUMBER: 94218359 MEDLINE
DOCUMENT NUMBER: 94218359 PubMed ID: 1344818
TITLE: Patterns of basement membrane laminin distribution in nonneoplastic and neoplastic thyroid tissue.
AUTHOR: Campo E; Perez M; Charonis A A; Axiotis C A; Merino M J
CORPORATE SOURCE: Laboratory of Pathology, National Institutes of Health, Bethesda, Maryland.
SOURCE: MODERN PATHOLOGY, (1992 Sep) 5 (5) 540-6.
Journal code: 8806605. ISSN: 0893-3952.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199405
ENTRY DATE: Entered STN: 19940606
Last Updated on STN: 19940606

Entered Medline: 19940524

AB Laminin, a major basement membrane component, is typically absent or partially lost around the epithelial elements of most invasive carcinomas. To evaluate the distribution of laminin in both primary and metastatic thyroid tumors, we studied 14 benign thyroid lesions (eight adenomas, two Graves' disease, two Hashimoto's thyroiditis, one adenomatous hyperplasia, one nodular goiter), 20 carcinomas (seven papillary, six tall cell variant, four follicular, three Hurthle), and eight metastases (five tall cell variant, three follicular) utilizing a polyclonal **antibody** against highly purified, **nidogen**-free laminin. All benign lesions showed positive, linear immunostaining along basement membranes. Partial loss or absence of laminin was seen in the solid areas of all types of thyroid carcinomas examined; well-differentiated papillary and follicular tumors, as well as papillary and follicular areas of more poorly differentiated neoplasms, maintained linear laminin immunostaining in the papillary cores beneath the epithelial cells and around follicles. A similar correlation between laminin deposition and architectural organization was seen in metastatic lesions. Hurthle cell carcinomas had a unique fragmented, pericellular immunostaining pattern around individual tumor cells, suggesting uncontrolled laminin synthesis. Our findings suggest that preservation of laminin production in thyroid tumors reflects their degree of differentiation and that absence of laminin correlates with lack of structural organization rather than reflecting invasive and metastatic potential.

L9 ANSWER 15 OF 29 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.DUPLICATE 12

ACCESSION NUMBER: 1993:95611 BIOSIS
DOCUMENT NUMBER: PREV199395050807
TITLE: Characterization of a natural human antibody with anti-galactosyl(alpha-1-2)galactose specificity that is present at high titers in chronic Trypanosoma cruzi infection.
AUTHOR(S): Avila, Jose Luis; Rojas, Miguel; Velaquez-Avila, Gladys
CORPORATE SOURCE: Inst. Biomed. Caracas, Venezuela, Hosp. de Ninos J. M. de los Rios, Caracas Venezuela
SOURCE: American Journal of Tropical Medicine and Hygiene, (1992) Vol. 47, No. 4, pp. 413-421.
ISSN: 0002-9637.
DOCUMENT TYPE: Article
LANGUAGE: English

AB An antibody reactive with the galactosyl(alpha-1-2)galactose (gal(alpha-1-2)gal) epitope was characterized in human sera by enzyme-linked immunosorbent assay, red blood cell (RBC) and laminin absorption, and oligosaccharide inhibition. This antibody was found evenly distributed between the IgG and IgM classes and was present at high titers in the serum of all normal adults studied, but in 75% of children less than three years of age, it was observed at the lower limit of detection, and gradually increased to adult levels by the age of six. Although this antibody bound to gal(alpha-1-3)gal-linked synthetic antigens, it did not bind to the same residues present in rabbit, rat, and guinea pig RBC or in murine laminin or **nidogen**. These latter results, plus the fact that antigen-**antibody** binding was strongly blocked by gal(alpha-1-2)gal but not by methyl-alpha-galactopyranoside or melibiose, suggest that this antibody is indeed different from anti-gal(alpha-1-3)gal antibody. Anti-gal(alpha-1-2)gal antibody levels were significantly elevated in 66% of patients with chronic chagasic cardiomyopathy, but were not elevated in patients with different clinical forms of leishmaniasis, Trypanosoma rangeli-infected patients, or in patients with 15 other infectious and inflammatory diseases. Gal(alpha-1-2)gal antibodies did not absorb to intact T. cruzi parasites, but absorbed strongly to trypomastigote and epimastigote sonicates, suggesting some masking of reactive epitopes. Since antibody binding is blocked by gal(alpha-1-3)gal, previous results suggest that in chronic T. cruzi infection, at least

three different antibody clones exist that react with gal(alpha-1-3)gal epitopes: anti-gal(alpha-1-3)gal IgG, anti-mannose (man)(alpha-1-3)gal or anti-man(beta-1-3)gal IgM, and anti-gal(alpha-1-2)gal IgM and IgG.

L9 ANSWER 16 OF 29 MEDLINE DUPLICATE 13
ACCESSION NUMBER: 92111677 MEDLINE
DOCUMENT NUMBER: 92111677 PubMed ID: 1370418
TITLE: American Leishmania spp. and Trypanosoma cruzi: galactosyl alpha(1-3) galactose epitope localization by colloidal gold immunocytochemistry and lectin cytochemistry.
AUTHOR: Bretana A; Avila J L; Contreras-Bretana M; Tapia F J
CORPORATE SOURCE: Secci+5Uon de Microscopia Electronica, Instituto de Biomedicina, Caracas, Venezuela.
SOURCE: EXPERIMENTAL PARASITOLOGY, (1992 Feb) 74 (1) 27-37. Journal code: 0370713. ISSN: 0014-4894.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199202
ENTRY DATE: Entered STN: 19920308
Last Updated on STN: 19960129
Entered Medline: 19920218

AB Patients with Chagas' disease or different clinical forms of leishmaniasis (cutaneous or visceral) have elevated galactosyl alpha (1-3)galactose antibodies. Using colloidal gold immunocytochemistry--monoclonal antibody gal-13 (specific for lipid-linked galactosyl alpha (1-3)galactose residues) and anti-**nidogen antibodies** and lectin cytochemistry (Bandeiraea simplicifolia IB4), both techniques specific for demonstrating galactosyl alpha (1-3)galactose residues--we have found terminal disaccharide residues on the Trypanosoma cruzi external surface of Vero cell-derived trypomastigotes but not in intact epimastigotes (although disrupted epimastigotes strongly stained), in the lips of the flagellar pocket, and on the parasitic side exactly opposite to the flagellar pocket in amastigote and promastigote forms of American Leishmania. These results resemble those obtained using anti-laminin antibodies in both trypanosomatids. In addition, results obtained with anti-**nidogen antibodies** seem to recognize in Trypanosoma cruzi and American Leishmania culture forms another different unknown terminal disaccharide. These results confirm the presence of terminal galactosyl alpha (1-3)galactose residues in both trypanosomatids, and that rabbit anti-laminin antibodies are indeed also recognizing galactosyl alpha (1-3)galactose residues as demonstrated for human circulating antibody. The presence of abundant galactosyl alpha (1-3)galactose residues on Trypanosomatid family members suggests a specific unknown role in parasite physiology for this terminal disaccharide.

L9 ANSWER 17 OF 29 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.DUPLICATE 14
ACCESSION NUMBER: 1990:518268 BIOSIS
DOCUMENT NUMBER: BA90:135544
TITLE: ULTRASTRUCTURAL LOCALIZATION OF THE CORE PROTEIN OF A BASEMENT MEMBRANE-SPECIFIC CHONDROITIN SULFATE PROTEOGLYCAN IN ADULT RAT SKIN.
AUTHOR(S): MCCARTHY K J; HORIGUCHI Y; COUCHMAN J R; FINE J-D
CORPORATE SOURCE: DEP. CELL BIOL. AND ANAT., VH 201 C BOX 803, UNIV. ALA. BIRMINGHAM, BIRMINGHAM, ALA. 35294, USA.
SOURCE: ARCH DERMATOL RES, (1990) 282 (6), 397-401. CODEN: ADREDL. ISSN: 0340-3696.
FILE SEGMENT: BA; OLD
LANGUAGE: English

AB Basement membranes are complex extracellular matrices present at epithelial/mesenchymal interfaces of tissues. The dermal-epidermal

junction has been shown to contain numerous components, some of the most well known being laminin, types IV and VII collagens, heparin sulfate proteoglycan, fibronectin, and entactin/**nidogen**. In this paper we show, using core protein-specific **antibodies**, the presence of a newly described basement membrane-specific chondroitin sulfate proteoglycan at the epithelial/mesenchmal interval of adult rat skin. Ultrastructurally, this antigen was proven to reside primarily within the basal lamina, apparently concentrated in the lamina densa. In addition, some of the proteoglycan was also present beneath the lamina densa, associated with the reticular lamina collagen fibrils.

L9 ANSWER 18 OF 29 MEDLINE DUPLICATE 15
ACCESSION NUMBER: 90384093 MEDLINE
DOCUMENT NUMBER: 90384093 PubMed ID: 2119467
TITLE: Entactin: a possible auto-antigen in the pathogenesis of non-Goodpasture anti-GBM nephritis.
AUTHOR: Saxena R; Bygren P; Butkowski R; Wieslander J
CORPORATE SOURCE: Department of Nephrology, University Hospital of Lund, Sweden.
SOURCE: KIDNEY INTERNATIONAL, (1990 Aug) 38 (2) 263-72. Journal code: 0323470. ISSN: 0085-2538.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199010
ENTRY DATE: Entered STN: 19901122
Last Updated on STN: 19980206
Entered Medline: 19901026

AB It has recently been demonstrated that many patients with various types of glomerulonephritis have antibodies to the 6M guanidine-HCl extract of glomerular basement membrane (Bygren et al, Nephrol Dial Transplant 4:254-261, 1989). In the present study a 150 K protein was isolated from the guanidine extract of bovine glomerular basement membrane utilizing ion exchange and gel filtration chromatographic procedures. Amino acid analysis and size of the isolated protein revealed similarity to that of entactin/nidogen. The identity of this protein as entactin/**nidogen** was further suggested by its precipitation with two different **antibodies** in a radioimmunoassay and by its reaction with four different antibodies in a sandwich ELISA. Inhibition of the antibodies to 150 K by bovine entactin, which was isolated separately and sequenced for amino acids, confirmed the identity of the 150 K protein as entactin/nidogen. Furthermore, it was shown that about one third of those patients who show antibodies to the crude guanidine extract have circulating antibodies directed against entactin. This was further confirmed by the competitive inhibition of antibodies to the crude guanidine extract in one of the positive serum by entactin in an ELISA inhibition and by immunoblotting experiments. These observations propose entactin as a possible non-Goodpasture glomerular basement membrane antigen that could be involved in the pathogenesis of certain forms of autoimmune glomerulonephritis (non-Goodpasture anti-GBM glomerulonephritis) in man. Most of these patients have a granular pattern of the immunoglobulin deposition along the glomerular basement membrane. This suggests the possibility that anti-GBM glomerulonephritis in human beings can have non-linear immunoglobulin deposits along the GBM.

L9 ANSWER 19 OF 29 MEDLINE DUPLICATE 16
ACCESSION NUMBER: 90118740 MEDLINE
DOCUMENT NUMBER: 90118740 PubMed ID: 2481931
TITLE: An improved immunofluorescence technique for the histological examination of blood vessel tissue.
AUTHOR: Kittelberger R; Davis P F; Stehbens W E
CORPORATE SOURCE: Malaghan Institute of Medical Research, Wellington School of Medicine, Wellington Hospital, New Zealand.

SOURCE: ACTA HISTOCHEMICA, (1989) 86 (2) 137-42.
Journal code: 0370320. ISSN: 0065-1281.
PUB. COUNTRY: GERMANY, EAST: German Democratic Republic
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199002
ENTRY DATE: Entered STN: 19900328
Last Updated on STN: 19960129
Entered Medline: 19900220

AB Autofluorescence of elastic fibres in blood vessel samples is a common interference with the specific fluorescence of FITC-conjugated antibodies. Counterstaining with eriochrome black T changed the yellow-green colour of elastic fibres to dark red, thus turning a disturbing feature into a useful reference background. A second counterstain, p-phenylenediamine, visualized cell nuclei as an amber colour. To demonstrate the improvement of this staining technique, cryosections from blood vessel samples, derived from control veins, arteries and experimental aneurysms of different ages (15 to 99 month old) in 5 sheep, were stained with **antibodies** against procollagen III, collagen type IV, laminin, and **nidogen**. The specific distribution of these connective tissue components could now be related to the location of the elastic fibres and the cells (cell nuclei).

L9 ANSWER 20 OF 29 MEDLINE
ACCESSION NUMBER: 90143096 MEDLINE
DOCUMENT NUMBER: 90143096 PubMed ID: 2482635
TITLE: [Structure and antigenicity of the glomerular basement membrane].
Aufbau und Antigenitat der glomerularen Basalmembran.
AUTHOR: Weber M
SOURCE: VERHANDLUNGEN DER DEUTSCHEN GESELLSCHAFT FUR PATHOLOGIE, (1989) 73 6-12. Ref: 38
Journal code: 7503704. ISSN: 0070-4113.
PUB. COUNTRY: GERMANY, WEST: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: German
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199003
ENTRY DATE: Entered STN: 19900328
Last Updated on STN: 19960129
Entered Medline: 19900312

AB The glomerular basement membrane is a complex extracellular matrix formed of various molecules which build a supramolecular network. The major structural components are collagen IV, laminin, heparan sulfate proteoglycan, and nidogen/entactin. Cross-reacting **antibodies** against laminin, **nidogen**, and collagen IV may occur after several infectious diseases. They are however of doubtful pathogenetic significance. The pathogenetic relevant autoantibodies in Goodpasture's syndrome and rapidly progressive glomerulonephritis with linear immunofluorescence pattern are directed against epitopes which are located on the collagenase resistant C-terminal globule NC1 of collagen IV. The human NC1 globule appears as a hexamer which dissociates into monomers and dimers under various experimental conditions. Dissociation is paralleled by a significant increase in available epitopes. Immunisation with the dissociated NC1 globule initiates a pulmo-renal syndrome in rabbits similar to the human Goodpasture's syndrome. In hereditary nephritis one of the alpha-chains which form the triple-helix of collagen IV seems to be altered within the NC1 region. This may possibly explain the typical morphologic findings in this disease as well as the reduced binding of antiglomerular basement membrane antibodies to basement membranes of kidneys in Alport's syndrome.

L9 ANSWER 21 OF 29 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1988:567788 CAPLUS
DOCUMENT NUMBER: 109:167788
TITLE: High resolution immunoelectron microscopic localization of functional domains of laminin, nidogen, and heparan sulfate proteoglycan in epithelial basement membrane of mouse cornea reveals different topological orientations
AUTHOR(S): Schittny, Johannes C.; Timpl, Rupert; Engel, Juergen
CORPORATE SOURCE: Biocent., Univ. Basel, Basel, CH-4056, Switz.
SOURCE: Journal of Cell Biology (1988), 107(4), 1599-610
CODEN: JCLBA3; ISSN: 0021-9525
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Thin and ultrathin cryosections of mouse cornea were labeled with affinity-purified antibodies directed against either laminin, its central segments (domain 1), the end of its long arm (domain 3), the end of one of its short arms (domain 4), nidogen, or low-d. heparan sulfate proteoglycan. All basement membrane proteins were detected by indirect immunofluorescence exclusively in the epithelial basement membrane, in Descemet's membrane, and in small amorphous plaques located in the stroma. Immunoelectron microscopy with the protein A-Au technique demonstrated laminin domain 1 and nidogen in a narrow segment of the lamina densa at the junction to the lamina lucida within the epithelial basement membrane. Domain 3 showed 3 preferred locations at both the cellular and stromal boundaries of the epithelial basement membrane and in its center. Domain 4 was located predominantly in the lamina lucida and the adjacent half of the lamina densa. The low-d. heparan sulfate proteoglycan was found all across the basement membrane, showing a similar uniform distribution as with antibodies against the whole laminin mol. In Descemet's membrane an even distribution was found with all these antibodies. Hence, within the epithelial basement membrane the center of the laminin mol. is located near the lamina densa/lamina lucida junction and its long arm favors 3 major orientations. One is close to the cell surface indicating binding to a cell receptor, whereas the other 2 are directed to internal matrix structures. The apparent codistribution of laminin domain 1 and nidogen agrees with biochem. evidence that nidogen binds to this domain.

L9 ANSWER 22 OF 29 MEDLINE DUPLICATE 17
ACCESSION NUMBER: 88151991 MEDLINE
DOCUMENT NUMBER: 88151991 PubMed ID: 3126070
TITLE: Analysis of degradation of the basement membrane protein **nidogen**, using a specific monoclonal **antibody**.
AUTHOR: Dziadek M; Clements R; Mitrangas K; Reiter H; Fowler K
CORPORATE SOURCE: Murdoch Institute for Research into Birth Defects, Royal Children's Hospital, Parkville, Victoria, Australia.
SOURCE: EUROPEAN JOURNAL OF BIOCHEMISTRY, (1988 Feb 15) 172 (1) 219-25.
Journal code: 0107600. ISSN: 0014-2956.
PUB. COUNTRY: GERMANY, WEST: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198804
ENTRY DATE: Entered STN: 19900308
Last Updated on STN: 19900308
Entered Medline: 19880419

AB A monoclonal **antibody** was produced against purified **nidogen** extracted from a mouse basement-membrane-producing tumor. This antibody reacted with a determinant on Nd-40, a rod which separates the globular domains of nidogen. Antigenicity depends on intrachain disulfide bonds within this rod. The monoclonal **antibody** was

used to detect **nidogen** fragments after proteolytic cleavage of isolated nidogen, and nidogen complexed to laminin. The data indicate that thrombin and thermolysin generated very different patterns of degradation, but in both cases no differences were found between isolated and complexed nidogen. In contrast, nidogen in the laminin-nidogen complex was much less degraded by trypsin than isolated nidogen, indicating that an interaction between these basement membrane components reduces the susceptibility of nidogen to trypsin digestion. Immunofluorescent studies, using the monoclonal antibody on sections of the EHS tumor after proteolytic digestion, showed that the retention or disappearance of the Nd-40 determinant correlated with the in vitro digestion pattern of the laminin-nidogen complex.

L9 ANSWER 23 OF 29 SCISEARCH COPYRIGHT 2003 ISI (R)
ACCESSION NUMBER: 88:109325 SCISEARCH
THE GENUINE ARTICLE: M2364
TITLE: ANALYSIS OF DEGRADATION OF THE BASEMENT-MEMBRANE PROTEIN **NIDOGEN**, USING A SPECIFIC MONOCLONAL-**ANTIBODY**
AUTHOR: DZIADEK M (Reprint); CLEMENTS R; MITRANGAS K; REITER H; FOWLER K
CORPORATE SOURCE: ROYAL CHILDRENS HOSP, MURDOCH INST RES BIRTH DEFECTS, PARKVILLE, VIC 3052, AUSTRALIA
COUNTRY OF AUTHOR: AUSTRALIA
SOURCE: EUROPEAN JOURNAL OF BIOCHEMISTRY, (1988) Vol. 172, No. 1, pp. 219-225.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: ENGLISH
REFERENCE COUNT: 42

L9 ANSWER 24 OF 29 MEDLINE DUPLICATE 18
ACCESSION NUMBER: 88139674 MEDLINE
DOCUMENT NUMBER: 88139674 PubMed ID: 2449451
TITLE: Serological activity against galactosyl-alpha(1-3)galactose in sera from patients with several kinetoplastida infections.
AUTHOR: Avila J L; Rojas M; Towbin H
CORPORATE SOURCE: Instituto de Biomedicina, Caracas, Venezuela.
SOURCE: JOURNAL OF CLINICAL MICROBIOLOGY, (1988 Jan) 26 (1) 126-32. Journal code: 7505564. ISSN: 0095-1137.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198804
ENTRY DATE: Entered STN: 19900308
Last Updated on STN: 19900308
Entered Medline: 19880407

AB Using rabbit erythrocyte-derived neutral glycosphingolipids enriched for a defined ceramide pentasaccharide as antigens, we have detected elevated anti-galactosyl-alpha(1-3)galactose (anti-G alpha G) antibody values in patients with American cutaneous leishmaniasis (ACL), chronic Chagas' disease, and Trypanosoma rangeli infections compared with normal subjects or with patients suffering from any of 15 other infectious diseases. The specificity of the G alpha G antibodies was determined by inhibition enzyme-linked immunosorbent assays, which revealed that several alpha-galactosyl- but not beta-galactosyl-bearing sugars blocked absorption of G alpha G antibodies to the specific antigen used. G alpha G antibodies were mainly distributed between immunoglobulin classes G and M in three Kinetoplastida infections studied, with a lower increase in reactivity detected in immunoglobulin A. Absorption of highly reactive G alpha G **antibodies** with purified murine laminin and **nidogen**, two basement membrane proteins, almost abolished G alpha

G reactivity, suggesting the identity of anti-G alpha G with laminin and **nidogen antibodies** previously reported as elevated in Kinetoplastida infections. In ACL, G alpha G antibodies were detected in 71% of patients having skin lesions with a clinical evolution time of 0.5 month. This percentage increased with the time of evolution of skin lesions, reaching 93% in lesions older than 3 months, and tended to decrease inversely to the induration diameter in the skin leishmanin test. It is proposed that similar epitopes may exist on kinetoplast protozoa and that the determination of G alpha G antibodies may be a highly sensitive assay for the detection of humoral responses to Kinetoplastida infections.

L9 ANSWER 25 OF 29 MEDLINE DUPLICATE 19
ACCESSION NUMBER: 87308118 MEDLINE
DOCUMENT NUMBER: 87308118 PubMed ID: 3114248
TITLE: The cellular interactions of laminin fragments. Cell adhesion correlates with two fragment-specific high affinity binding sites.
AUTHOR: Aumailley M; Nurcombe V; Edgar D; Paulsson M; Timpl R
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (1987 Aug 25) 262 (24) 11532-8.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198709
ENTRY DATE: Entered STN: 19900305
Last Updated on STN: 19970203
Entered Medline: 19870930

AB The molecular interactions of laminin with several tumor cell lines and skin fibroblasts were investigated by radioligand binding studies and cell attachment assays using laminin, the laminin-**nidogen** complex, and laminin fragments as substrates and also domain-specific **antibodies** as inhibitors of cell attachment. The majority of cells showed a dual binding pattern for fragments 1 and 8 which originate from short-arm or long-arm structures of laminin, respectively. Both of these fragments in solution bind to suspended cells with high affinity (KD = 1-10 nM), with the receptor numbers for each fragment depending on the cell type. Competition studies and independent variation of receptor numbers demonstrated that the cell-binding structures on each fragment are different, implicating the existence of two distinct cellular receptors for laminin. The ability of these fragments to act as substrates for cell adhesion correlated with the presence of high affinity binding sites on the cells. However, only antibodies to fragment 8 were able to block cell adhesion to laminin, despite the presence of binding sites for fragment 1. A few cells had very low numbers of high affinity receptors for either fragment 1 or 8. The latter cell type was used to demonstrate that complex formation between laminin and nidogen, which binds to fragment 1 structures, reduces the potential of laminin for cell binding.

L9 ANSWER 26 OF 29 MEDLINE DUPLICATE 20
ACCESSION NUMBER: 88136304 MEDLINE
DOCUMENT NUMBER: 88136304 PubMed ID: 2449305
TITLE: **Antibodies** to basement membrane proteins **nidogen** and laminin in sera from streptococcal-related diseases and juvenile rheumatoid arthritis patients.
AUTHOR: Avila J L; Rojas M; Velazquez-Avila G; Rieber M
CORPORATE SOURCE: Instituto de Biomedicina, Caracas, Venezuela.
SOURCE: CLINICAL AND EXPERIMENTAL IMMUNOLOGY, (1987 Dec) 70 (3) 555-61.
Journal code: 0057202. ISSN: 0009-9104.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198803
ENTRY DATE: Entered STN: 19900308
Last Updated on STN: 19900308
Entered Medline: 19880328

AB Using the ELISA technique, **antibodies** against two different basement proteins, laminin and **nidogen** (ALNA), were determined in 226 children suffering from one of 37 different inflammatory or infectious diseases. These included 80 patients with streptococcal infection and 40 with juvenile rheumatoid arthritis. Forty-eight percent of the streptococcus-infected patients (or 75% of those in the acute phase) and 60% of juvenile rheumatoid arthritis patients had significantly elevated ALNA levels compared with healthy controls. Interestingly 10 adult rheumatoid arthritis patients displayed normal ALNA levels, suggesting a particular immune process occurring in children affected by juvenile rheumatoid arthritis. By means of periodate oxidation and glycosidase treatments we have shown that ALNA positive sera recognized terminal alpha-galactose as the reactive epitope.

L9 ANSWER 27 OF 29 MEDLINE DUPLICATE 21
ACCESSION NUMBER: 87034242 MEDLINE
DOCUMENT NUMBER: 87034242 PubMed ID: 2429987
TITLE: **Antibodies** to basement membrane protein **nidogen** in Chagas' disease and American cutaneous leishmaniasis.
AUTHOR: Avila J L; Rojas M; Velazquez-Avila G; von der Mark H; Timpl R
SOURCE: JOURNAL OF CLINICAL MICROBIOLOGY, (1986 Nov) 24 (5) 775-8. Journal code: 7505564. ISSN: 0095-1137.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198612
ENTRY DATE: Entered STN: 19900302
Last Updated on STN: 19900302
Entered Medline: 19861216

AB About 50 to 70% of sera from patients with American cutaneous leishmaniasis and chronic Chagas' disease possessed **antibodies** which reacted in enzyme and radioimmunoassays with **nidogen** obtained from a tumor basement membrane. The antibodies were of the immunoglobulin M and G classes in acute American cutaneous leishmaniasis but mainly of the immunoglobulin G class in chronic Chagas' disease. Similar antibodies could not be detected in patients suffering from a variety of other infectious or inflammatory diseases when compared with healthy control groups. Inhibition and immunoadsorption studies indicated a close relationship of epitopes recognized by patients' **antibodies** on **nidogen** and on another basement membrane protein, laminin. Since rabbit antisera to both proteins do not cross-react, a special nature of the epitopes involved in the reaction with patient sera is suggested. Similar epitopes may exist on various forms of Leishmania or Trypanosoma protozoa.

L9 ANSWER 28 OF 29 MEDLINE DUPLICATE 22
ACCESSION NUMBER: 86005830 MEDLINE
DOCUMENT NUMBER: 86005830 PubMed ID: 2995165
TITLE: Expression of nidogen and laminin in basement membranes during mouse embryogenesis and in teratocarcinoma cells.
AUTHOR: Dziadek M; Timpl R
SOURCE: DEVELOPMENTAL BIOLOGY, (1985 Oct) 111 (2) 372-82. Journal code: 0372762. ISSN: 0012-1606.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198510
ENTRY DATE: Entered STN: 19900321
Last Updated on STN: 19900321
Entered Medline: 19851029

AB Nidogen and laminin were localized at preimplantation stages of mouse development by immunofluorescence. Laminin was already present on the cell surface at the 2-cell stage, while nidogen was first detectable on compacted 8- to 16-cell stage morulae. Nidogen and laminin colocalized at the blastocyst stage and in postimplantation basement membranes. Immunoblot analyses of tissue extracts and cell culture media indicated the 150-kDa form of nidogen as the largest and predominant form in all tissues examined. Radiolabeled **nidogen** and laminin synthesized by Reichert's membrane were coprecipitated by **antibodies** against each antigen, indicating complex formation in situ. Equimolar amounts of laminin and nidogen were determined in 6 M guanidine X HCl extracts of tissues by radioimmunoassays, further indicating stoichiometric complexes. However, lower levels of nidogen than laminin were found in tissue and cell culture media. A less than 2-fold increase in nidogen was found when F9 cells were stimulated to differentiate with retinoic acid and dibutyryl cAMP, compared to a 30-fold increase in laminin secretion.

L9 ANSWER 29 OF 29 MEDLINE DUPLICATE 23
ACCESSION NUMBER: 84108344 MEDLINE
DOCUMENT NUMBER: 84108344 PubMed ID: 6420150
TITLE: Nidogen: a new, self-aggregating basement membrane protein.
AUTHOR: Timpl R; Dziadek M; Fujiwara S; Nowack H; Wick G
SOURCE: EUROPEAN JOURNAL OF BIOCHEMISTRY, (1983 Dec 15) 137 (3) 455-65.
Journal code: 0107600. ISSN: 0014-2956.
PUB. COUNTRY: GERMANY, WEST: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198403
ENTRY DATE: Entered STN: 19900319
Last Updated on STN: 19900319
Entered Medline: 19840301

AB Nidogen was purified from a mouse tumor basement membrane where it accounted for 2-3% of the total proteins. It was isolated as two forms (A and B) of a monomer (Mr = 80000) each consisting of a single polypeptide chain folded into a globular head connected to a small tail. The B form of the monomer was shown to be capable of aggregating into a nest-like structure (Mr greater than 250000). A smaller form (Mr = 45000) was observed in some of the extracts. The amino acid composition of nidogen was different to that of other basement membrane proteins. It contained about 10% carbohydrate, with N-linked and O-linked oligosaccharide chains in similar proportions. Isoelectrofocussing demonstrated a limited heterogeneity of nidogen with pI in the range 6.5 - 7. Monomeric nidogen failed to interact with other basement membrane components and heparin. Aggregation could be induced by limited proteolysis and was reversed by detergents or high salt concentrations. Together with the observation that most of the nidogen could be solubilized only after destroying the collagenous matrix, the data indicate that aggregation of nidogen reflects an activity involved in matrix assembly. Specific **antibodies** raised against **nidogen** did not distinguish between the monomeric and aggregated form of the protein but showed that the fragment was antigenically deficient. These antibodies did not cross-react with collagen type IV, laminin, entactin and heparansulfate proteoglycan. Immunofluorescence staining and absorption studies demonstrated that nidogen is a common component of authentic basement membranes. Larger forms of nidogen (Mr about 100000 and 150000) were found in organ cultures of Reichert's membrane suggesting that it is synthesized in precursor

forms.

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                  ENERGY, INSPEC
 NEWS 20  Feb 13  CANCERLIT is no longer being updated
 NEWS 21  Feb 24  METADEX enhancements
 NEWS 22  Feb 24  PCTGEN now available on STN
 NEWS 23  Feb 24  TEMA now available on STN
 NEWS 24  Feb 26  NTIS now allows simultaneous left and right truncation
 NEWS 25  Feb 26  PCTFULL now contains images
 NEWS 26  Mar 04  SDI PACKAGE for monthly delivery of multifile SDI results
 NEWS 27  Mar 19  APOLLIT offering free connect time in April 2003
 NEWS 28  Mar 20  EVENTLINE will be removed from STN
 NEWS 29  Mar 24  PATDPAFULL now available on STN
 NEWS 30  Mar 24  Additional information for trade-named substances without
                  structures available in REGISTRY
 NEWS 31  Apr 11  Display formats in DGENE enhanced
 NEWS 32  Apr 14  MEDLINE Reload
 NEWS 33  Apr 17  Polymer searching in REGISTRY enhanced
 NEWS 34  Apr 21  Indexing from 1947 to 1956 being added to records in CA/CAPLUS
 NEWS 35  Apr 21  New current-awareness alert (SDI) frequency in
                  WPIDS/WPINDEX/WPIX

 NEWS EXPRESS  April 4 CURRENT WINDOWS VERSION IS V6.01a, CURRENT
               MACINTOSH VERSION IS V6.0b(ENG) AND V6.0Jb(JP),
               AND CURRENT DISCOVER FILE IS DATED 01 APRIL 2003
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 NEWS PHONE    Direct Dial and Telecommunication Network Access to STN
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=> file medline, cancerlit, biosis, confsci, caplus, embase, uspatfull, pctfull,
scisearch
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                                                         ENTRY    SESSION
FULL ESTIMATED COST                                       0.21       0.21

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=> s nidogen and antibod?
L1          713 NIDOGEN AND ANTIBOD?

=> dup rem l1
PROCESSING COMPLETED FOR L1
L2          407 DUP REM L1 (306 DUPLICATES REMOVED)

=> s l2 not py=>2000
'2000' NOT A VALID FIELD CODE
   7 FILES SEARCHED...
L3          244 L2 NOT PY=>2000

=> d l-10

L3   ANSWER 1 OF 244      MEDLINE
AN   2000004632      MEDLINE
```

DN 20004632 PubMed ID: 10533057
TI Suppression of **nidogen**-1 translation by antisense targeting affects the adhesive properties of cultured astrocytes.
AU Grimpe B; Probst J C; Hager G
CS Department of Neuromorphology, Max-Planck-Institute of Neurobiology, Martinsried, Germany.
SO GLIA, (1999 Nov) 28 (2) 138-49.
Journal code: 8806785. ISSN: 0894-1491.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199912
ED Entered STN: 20000113
Last Updated on STN: 20000113
Entered Medline: 19991215

L3 ANSWER 2 OF 244 MEDLINE
AN 1999359536 MEDLINE
DN 99359536 PubMed ID: 10398106
TI Renal cell carcinomas and pancreatic adenocarcinomas produce **nidogen** in vitro and in vivo.
AU Oivula J; Lohi J; Tani T; Kangas L; Kiviluoto T; Kivilaakso E; Butkowski R; Virtanen I
CS Department of Anatomy, Institute of Biomedicine, P.O. Box 9 (Siltavuorenpenger 20A), FIN-00014 University of Helsinki, Helsinki, Finland.. Jan.Oivula@helsinki.fi
SO JOURNAL OF PATHOLOGY, (1999 Mar) 187 (4) 455-61.
Journal code: 0204634. ISSN: 0022-3417.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199908
ED Entered STN: 19990827
Last Updated on STN: 19990827
Entered Medline: 19990817

L3 ANSWER 3 OF 244 MEDLINE
AN 1999343071 MEDLINE
DN 99343071 PubMed ID: 10416682
TI Hemidesmosomal molecular changes in dermatitis herpetiformis; decreased expression of BP230 and plectin/HD1 in uninvolved skin.
AU Leivo T; Lohi J; Kariniemi A L; Molander G; Kiraly C L; Kotovirta M L; Owaribe K; Burgeson R E; Leivo I
CS Department of Anatomy, Institute of Biomedicine, University of Helsinki, Finland.
SO HISTOCHEMICAL JOURNAL, (1999 Feb) 31 (2) 109-16.
Journal code: 0163161. ISSN: 0018-2214.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199908
ED Entered STN: 19990827
Last Updated on STN: 19990827
Entered Medline: 19990818

L3 ANSWER 4 OF 244 MEDLINE
AN 1999242614 MEDLINE
DN 99242614 PubMed ID: 10225960
TI Characterization and expression of the laminin gamma3 chain: a novel, non-basement membrane-associated, laminin chain.
AU Koch M; Olson P F; Albus A; Jin W; Hunter D D; Brunken W J; Burgeson R E; Champliaud M F
CS The Cutaneous Biology Research Center, Massachusetts General Hospital, and the Department of Dermatology, Harvard Medical School, Charlestown, Massachusetts 02129, USA.
NC AR35689 (NIAMS)
SO JOURNAL OF CELL BIOLOGY, (1999 May 3) 145 (3) 605-18.
Journal code: 0375356. ISSN: 0021-9525.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-AF041835
EM 199906
ED Entered STN: 19990614
Last Updated on STN: 19990614
Entered Medline: 19990601

L3 ANSWER 5 OF 244 MEDLINE
AN 1999125843 MEDLINE
DN 99125843 PubMed ID: 9928763
TI Immunohistochemical study on phenotypical changes of hepatocytes in liver disease with reference to extracellular matrix composition.
AU Matsumoto S; Yamamoto K; Nagano T; Okamoto R; Ibuki N; Tagashira M; Tsuji T
CS First Department of Internal Medicine, Okayama University Medical School, Japan.
SO LIVER, (1999 Feb) 19 (1) 32-8.
Journal code: 8200939. ISSN: 0106-9543.
CY Denmark
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199903
ED Entered STN: 19990402
Last Updated on STN: 19990402
Entered Medline: 19990324

L3 ANSWER 6 OF 244 MEDLINE
AN 1999116367 MEDLINE
DN 99116367 PubMed ID: 9917842
TI Laminin isoforms and epithelial development.
AU Ekblom M; Falk M; Salmivirta K; Durbeej M; Ekblom P
CS Department of Animal Physiology, Uppsala University, Sweden.
SO ANNALS OF THE NEW YORK ACADEMY OF SCIENCES, (1998 Oct 23) 857 194-211.
Ref: 68
Journal code: 7506858. ISSN: 0077-8923.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals
EM 199902
ED Entered STN: 19990301
Last Updated on STN: 19990301
Entered Medline: 19990216

L3 ANSWER 7 OF 244 MEDLINE
AN 1999049433 MEDLINE
DN 99049433 PubMed ID: 9833687
TI Basal lamina molecules are concentrated in myogenic regions of the mouse limb bud.
AU Godfrey E W; Gradall K S
CS Department of Cell Biology, Neurobiology and Anatomy, Medical College of Wisconsin, Milwaukee 53225, USA.. egodfrey@mcw.edu
NC HD20743 (NICHD)

SO ANATOMY AND EMBRYOLOGY, (1998 Dec) 198 (6) 481-6.
Journal code: 7505194. ISSN: 0340-2061.
CY GERMANY: Germany, Federal Republic of
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199901
ED Entered STN: 19990216
Last Updated on STN: 19990216
Entered Medline: 19990129

L3 ANSWER 8 OF 244 MEDLINE
AN 1998436084 MEDLINE
DN 98436084 PubMed ID: 9765055
TI Phenotypical changes of a human pancreatic adenocarcinoma cell line after selection on laminin-1/**nidogen** (LM/Ng) substratum.
AU Paddenberg R; Flocke K; Elsasser H P; Lesch G; Heidtmann H H; Mannherz H G
CS Department of Anatomy and Embryology, Ruhr-University Bochum, Germany.
SO EUROPEAN JOURNAL OF CELL BIOLOGY, (1998 Aug) 76 (4) 251-64.
Journal code: 7906240. ISSN: 0171-9335.
CY GERMANY: Germany, Federal Republic of
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199812
ED Entered STN: 19990115
Last Updated on STN: 20000303
Entered Medline: 19981211

L3 ANSWER 9 OF 244 MEDLINE
AN 1998406162 MEDLINE
DN 98406162 PubMed ID: 9733643
TI **Nidogen**-2: a new basement membrane protein with diverse binding properties.
AU Kohfeldt E; Sasaki T; Gohring W; Timpl R
CS Max-Planck-Institut fur Biochemie, D-82152 Martinsried, Germany.
SO JOURNAL OF MOLECULAR BIOLOGY, (1998 Sep 11) 282 (1) 99-109.
Journal code: 2985088R. ISSN: 0022-2836.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-AJ223500
EM 199810
ED Entered STN: 19981021
Last Updated on STN: 19981021
Entered Medline: 19981015

L3 ANSWER 10 OF 244 MEDLINE
AN 1998402596 MEDLINE
DN 98402596 PubMed ID: 9731120
TI Laminin heterogeneity around Schlemm's canal in normal humans and glaucoma patients.
AU Dietlein T S; Jacobi P C; Paulsson M; Smyth N; Krieglstein G K
CS Department of Ophthalmology, Medical Faculty, University of Cologne, Germany.
SO OPHTHALMIC RESEARCH, (1998) 30 (6) 380-7.
Journal code: 0267442. ISSN: 0030-3747.
CY Switzerland
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199901
ED Entered STN: 19990128
Last Updated on STN: 19990128
Entered Medline: 19990108

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(FILE 'HOME' ENTERED AT 13:34:50 ON 24 APR 2003)

FILE 'MEDLINE, CANCERLIT, BIOSIS, CONFSCI, CAPLUS, EMBASE, USPATFULL, PCTFULL, SCISEARCH' ENTERED AT 13:35:24 ON 24 APR 2003
L1 713 S NIDOGEN AND ANTIBOD?
L2 407 DUP REM L1 (306 DUPLICATES REMOVED)
L3 244 S L2 NOT PY=>2000

=> s l3 and fusion
L4 45 L3 AND FUSION

=> s l4 and humanize?
L5 10 L4 AND HUMANIZE?

=> s l5 and single (a) chain
L6 9 L5 AND SINGLE (A) CHAIN

=> d 1-9

L6 ANSWER 1 OF 9 USPATFULL
AN 1999:117652 USPATFULL
TI Zinc binding LIM protein S2-6
IN Lecka-Czernik, Beata, 8710 Boulder La., Little Rock, AR, United States 72227
PA The Board of Trustees of the University of Arkansas, Little Rock, AR, United States (U.S. corporation)
PI US 5959081 19990928
AI US 1997-856444 19970514 (8)
DT Utility
FS Granted
LN.CNT 2172
INCL INCLM: 530/358.000
INCLS: 435/320.100; 435/325.000; 536/023.100
NCL NCLM: 530/358.000
NCLS: 435/320.100; 435/325.000; 536/023.100
IC [6]
ICM: A61K038-16
ICS: C12N015-00; C12N005-00; C07H021-02
EXF 435/320.1; 435/325; 530/358; 536/23.1
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 2 OF 9 USPATFULL
AN 1999:110213 USPATFULL
TI Compositions for the diagnosis and treatment of Chediak-Higashi syndrome
IN Kaplan, Jerry, Salt Lake City, UT, United States
Perou, Charles M., Salt Lake City, UT, United States
Moore, Karen J., Maynard, MA, United States
PA Millennium Pharmaceuticals, Cambridge, MA, United States (U.S. corporation)
The University of Utah Research Foundation, Salt Lake City, UT, United States (U.S. corporation)
PI US 5952223 19990914
AI US 1997-822445 19970321 (8)
DT Utility
FS Granted
LN.CNT 5033
INCL INCLM: 435/325.000
INCLS: 435/006.000; 435/320.100; 536/023.500
NCL NCLM: 435/325.000
NCLS: 435/006.000; 435/320.100; 536/023.500

IC [6]
ICM: C12N015-85
EXF 536/23.5; 536/23.51; 435/6; 435/172.3; 435/69.1; 435/325; 435/320.1
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 3 OF 9 USPATFULL
AN 1999:59136 USPATFULL
TI DNA binding protein S1-3
IN Lecka-Czernik, Beata, Little Rock, AR, United States
PA University of Arkansas, Little Rock, AR, United States (U.S. corporation)
PI US 5905146 19990518
AI US 1996-616857 19960315 (8)
DT Utility
FS Granted
LN.CNT 3236
INCL INCLM: 536/023.500
INCLS: 435/252.300; 435/320.100; 435/325.000; 536/024.310
NCL NCLM: 536/023.500
NCLS: 435/252.300; 435/320.100; 435/325.000; 536/024.310
IC [6]
ICM: C07H021-04
ICS: C12N001-21; C12N015-12; C12N015-85
EXF 536/24.31; 536/23.1; 536/23.5; 435/320.1; 435/240.1; 435/6; 435/325; 435/252.3
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 4 OF 9 PCTFULL COPYRIGHT 2003 Univentio
AN 1999066959 PCTFULL ED 20020515
TIEN COMPOSITIONS AND METHODS FOR DELIVERY OF AGENTS FOR ALTERING NEURONAL GROWTH, REGENERATION, AND SURVIVAL
TIFR COMPOSITIONS ET PROCEDES PERMETTANT LA MISE EN PLACE D'AGENTS VENANT MODIFIER LA CROISSANCE DES NEURONES, LEUR REGENERATION ET LEUR SURVIE
IN BAIRD, Andrew;
BERRY, Martin;
LOGAN, Ann;
GONZALEZ, Ana Maria
PA SELECTIVE GENETICS, INC.;
BAIRD, Andrew;
BERRY, Martin;
LOGAN, Ann;
GONZALEZ, Ana Maria
LA English
DT Patent
PI WO 9966959 A2 19991229
DS W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG US UZ VN YU ZA ZW GH GM KE LS MW SD SL SZ UG ZW AM AZ BY KG KZ MD RU TJ TM AT BE CH CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE BF BJ CF CG CI CM GA GN GW ML MR NE SN TD TG
AI WO 1999-US12126 A 19990601
PRAI US 1998-09/088,419 19980601
US 1998-09/178,286 19981023
ICM A61K048-00

L6 ANSWER 5 OF 9 PCTFULL COPYRIGHT 2003 Univentio
AN 1999016882 PCTFULL ED 20020515
TIEN i(LMB) GENE OF i(STREPTOCOCCUS AGALACTIAE)
TIFR GENE i(LMB) DE i(STREPTOCOCCUS AGALACTIAE)
IN SPELLERBERG, Barbara;
LUTTICKEN, Rudolf;
PODBIELSKI, Andreas;
ROZDZINSKI, Eva
PA MEDIMMUNE, INC.
LA English
DT Patent
PI WO 9916882 A1 19990408
DS W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GD GE GH GM HU ID IL IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG UZ VN YU ZW GH GM KE LS MW SD SZ UG ZW AM AZ BY KG KZ MD RU TJ TM AT BE CH CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE BF BJ CF CG CI CM GA GN GW ML MR NE SN TD TG
AI WO 1998-US20028 A 19980925
PRAI US 1997-60/059,952 19970926
ICM C12N015-31
ICS C12N015-12; C12N001-21; C07K014-315; A61K039-09; C07K016-12; C12N001-21; C12R001-46

L6 ANSWER 6 OF 9 PCTFULL COPYRIGHT 2003 Univentio
AN 1998012306 PCTFULL ED 20020514
TIEN HEMATOPOIETIC STEM CELLS AND METHODS FOR GENERATING SUCH CELLS
TIFR CELLULES SOUCHES HEMATOPOIETIQUES ET PROCEDES RELATIFS A LEUR PRODUCTION
IN CARLSSON, Leif
PA ONTOGENY, INC.;
CARLSSON, Leif
LA English
DT Patent
PI WO 9812306 A1 19980326
DS W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GE HU IL IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG US UZ VN ZW GH KE LS MW SD SZ UG ZW AM AZ BY KG KZ MD RU TJ TM AT BE CH DE DK ES FI FR GB GR IE IT LU MC NL PT SE BF BJ CF CG CI CM GA GN ML MR NE SN TD TG
AI WO 1997-IB1246 A 19970923
PRAI US 1996-8/717,585 19960923
ICM C12N005-08
ICS C12N005-10; A61K048-00; C07K014-47; A61K038-17

L6 ANSWER 7 OF 9 PCTFULL COPYRIGHT 2003 Univentio
AN 1997034914 PCTFULL ED 20020514
TIEN COMPOSITIONS FOR THE DIAGNOSIS AND TREATMENT OF CHEDIAK-HIGASHI SYNDROME
TIFR COMPOSITIONS DESTINEES AU DIAGNOSTIC ET AU TRAITEMENT DU SYNDROME DE CHEDIAK-HIGASHI
IN KAPLAN, Jerry;
PEROU, Charles, M.;
MOORE, Karen, J.
PA MILLENNIUM PHARMACEUTICALS, INC.;
UNIVERSITY OF UTAH
LA English
DT Patent
PI WO 9734914 A1 19970925
DS W: AU CA JP AT BE CH DE DK ES FI FR GB GR IE IT LU MC NL PT SE
AI WO 1997-US5068 A 19970321
PRAI US 1996-60/013,883 19960322
US 1996-60/015,673 19960419
US 1996-60/021,064 19960701
ICM C07H021-04
ICS C07K014-435; C07K016-18; C12Q001-68; G01N033-53; C12N015-63; A61K048-00

L6 ANSWER 8 OF 9 PCTFULL COPYRIGHT 2003 Univentio
AN 1997028262 PCTFULL ED 20020514
TIEN LYST1 AND LYST2 GENE COMPOSITIONS AND METHODS OF USE
TIFR COMPOSITIONS DE GENES LYST1 ET LYST2 ET LEURS PROCEDES D'UTILISATION
IN KINGSMORE, Stephen, F.;

BARBOSA-ALLEYNE, Maria, D., F., S.
PA UNIVERSITY OF FLORIDA;
KINGSMORE, Stephen, F.;
BARBOSA-ALLEYNE, Maria, D., F., S.
LA English
DT Patent
PI WO 9728262 A1 19970807
DS W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI
GB GE HU IL IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG
MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK TJ TM TR TT UA
UG US US US UZ VN KE LS MW SD SZ UG AM AZ BY KG KZ MD RU TJ
TM AT BE CH DE DK ES FI FR GB GR IE IT LU MC NL PT SE BF BJ
CF CG CI CM GA GN ML MR NE SN TD TG
AI WO 1997-US1748 A 19970131
PRAI US 1996-60/011,146 19960201
US 1996-60/033,599 19961220
US 1996-60/034,346 19961223
ICM C12N015-12
ICS C07K014-47; C12N005-10; C07K016-18; A01K067-027

L6 ANSWER 9 OF 9 PCTFULL COPYRIGHT 2003 Univentio
AN 1995017673 PCTFULL ED 20020514
TIEN OCULAR DIAGNOSTICS AND THERAPIES
TIFR DIAGNOSTICS ET THERAPIES OCULAIRES
IN HAGEMAN, Gregory, S.
PA OCUTECH, INC.;
HAGEMAN, Gregory, S.
LA English
DT Patent
PI WO 9517673 A1 19950629
DS W: AM AT AU BB BG BR BY CA CH CN CZ DE DK EE ES FI GB GE HU JP
KE KG KP KR KZ LK LR LT LU LV MD MG MN MW NL NO NZ PL PT RO
RU SD SE SI SK TJ TT UA US UZ VN KE MW SD SZ AT BE CH DE DK
ES FR GB GR IE IT LU MC NL PT SE BF BJ CF CG CI CM GA GN ML
MR NE SN TD TG
AI WO 1994-US14691 A 19941220
PRAI US 1993-8/172,129 19931221
ICM G01N033-533
ICS G01N033-536; G01N033-544; G01N033-558; G01N033-563; G01N033-566;
A61K039-395; A61K051-00; A61K051-08; A61K051-10; A61K051-12

=> d kwic 9

L6 ANSWER 9 OF 9 PCTFULL COPYRIGHT 2003 Univentio

DETD Immunoc[lobulins
The invention includes immunoglobulins, especially noglobulins directed against vitronectin. Immunoglobulins or **antibodies** are proteins that bind to an antigen. As used herein,, the term immunoglobulin or **antibody** refers to an entire immunoglobulin or **antibody** or any functional fragment of an immunoglobulin molecule. Examples include complete antibo'dy molecules, **antibody** fragments, such as Fab, F(abl)2, CDRsj VLAP VH, and any other portion of an **antibody**.

Immunoglobulins are typically composed of four covalently bound peptide chains. For example, an IgG **antibody** has two light chains and two heavy chains. Each light chain is covalently bound to a heavy chain. In turn each heavy chain. . .
. . .
molecules, or even heavy or light chains alone, may bind antigen. As used herein, vitronectin or fragments thereof can be an antigen. **Antibodies**, fragments of **antibodies**, and individual chains are all referred to herein as immunoglobulins.

A normal **antibody** heavy or light chain has an N-terminal (NH2) variable (V) region, and a C-terminal (-COOH) constant (C) region. The heavy chain. . . is referred to as VL (including V. or
V,). The variable region is the part of the molecule that binds to the **antibody**'s cognate antigen, while the Fc region (the second and third domains of the C region) determines the **antibody**'s effector function (e.g., complement fixation, opsonization).
. .
The sequences of the framework regions of different light or heavy chains are relatively conserved within a species. The framework region of an **antibody**, that is the combined framework regions of the constituent light and heavy chains, serves to position and align the CDRs in three. . .

Likewise, the constant region of the heavy chain molecule, also known as CHF determines the isotype of the **antibody**, **Antibodies** are referred to as IgM, IgDj IgG, IgA, and IgE depending on the heavy chain isotype. The isotypes are encoded in the. . .

The heavy chain isotypes determine different effector functions of the **antibody**. In addition, the heavy chain isotype determines the secreted form of the **antibody**.

Immunoglobulins are frequently classified according to their valency, IgG is a bivalent **antibody** and IgM is a polyvalent **antibody**. The valency refers to the number of binding sites on the immunoglobulin. Monovalent means that one **antibody** molecule binds to one receptor, bivalent means that the **antibody** binds to exactly two receptors and polyvalent or multivalent means that it binds to two or more receptors. Polyclonal **antibodies** generally comprise a mixture of bivalent **antibodies**.

Methods of the invention relating to vitronectin binding molecules include the use of monovalent immunoglobulins. In preferred embodiments, a single monovalent monoclonal **antibody** or a mixture of monovalent **antibodies**, such as two or more monoclonal **antibodies**
, is
used. A particular embodiment is a monospecific (as monoclonal **antibodies** are by definition), monovalent (i.e Fab fragment or **single chain antibody**)
monoclonal probe.

The immunoglobulins can be prepared in a variety of ways known in the art, depending upon whether monoclonal or polyclonal **antibodies** are desired. For polyclonal **antibodies**,
a vertebrate, typically a domestic animal, is hyperimmunized with the antigen, blood from the vertebrate is collected shortly after immunization and the gamma. . .

Suitable methods for preparing polyclonal **antibodies** are described in the Handbook of Experimental Immunology, 3d edel Weir (ed.), Blackwell Scientific Publications (1978).

For monoclonal **antibodies**, a small animal, typically a rat or mouse, is hyperimmunized with antigen, the spleen is

removed and the lymphocytes are fused with myeloma cells in the presence of a suitable **fusion** promoter. The resulting hybrid cells or hybridomas are screened to isolate individual clones, each of which secrete a single **antibody** species to the antigen. The individual **antibody** species are each the product of a single B cell generated in response to a specific antigenic site recognized on the antigen or immunogenic substance. The process for obtaining monoclonal **antibodies** is described by Kohler and Milstein, Nature, 256:495 (1975)o See also Harlow and Lane, **Antibodies**: A Laboratory Manual, Cold Spring Harbor Publications, N.Y. (1988)e
The peptides or antigens used to generate the **antibodies**, depending upon their own immunogenicity, may be used directly in the immunization procedure as immunogenic components associated with living or fixed cells. . .

. . .
also to DNA sequences. The DNA sequences associated with this invention include, for example, DNA subsequences encoding amino acid sequences of the **antibody** heavy or light chains, or fragments thereof, which determine binding specificity for a vitronectin receptor protein. These sequences may be ligated, for example,. . . into human constant region expression vectors, and inserted into a host cell. The host cell can then express a recombinant chimeric or hybrid **antibody** that is.specific for binding to a vitronectin receptor protein or polypeptide.

. . .
kDa and contains a single antigen binding site. Fab fragments may be obtained from F(abl)2 fragments by limited reduction, or from whole **antibody** by digestion with papain in the presence of reducing agents (see Harlow and Lane, supra).

Chimeric **antibodies** may also be used in this invention. Chimeric **antibodies** or chimeric peptides refer to those **antibodies** or **antibody** peptides wherein one portion
of the peptide has an amino acid sequence that is derived from, or is homologous to, a corresponding sequence in an **antibody** or peptide derived from a first gene source, while the remaining segment of the chain(s) is homologous to corresponding sequences of another gene source. For example, a chimeric **antibody** peptide may comprise an **antibody** heavy
chain with a murine variable region and a human constant region. The two gene sources will typically involve two species, but will. . .

An example of a successful human/murine chimeric **antibody** is one for carcinoembryonic (CEA) antigen described by Beidler, et al., J. of Immunology, 141:4053 (1988). Other methods for constructing chimeric **antibodies** and binding fragments are described in Brown et al., Cancer Research 47:3577-3583 (1987); Kameyama et al., FEB 2:301-306 (1989); Orlandi et al,, PNAS. . . al.,
Clin. Chem. 34:1668-1675 (1988); Better et al,, Science 240:1041 (1988) and Morrison and Oi, Advances in Immunology 44:55 (1989)e
More broadly, a chimeric **antibody** is any **antibody** in which either or both of the heavy or light chains are composed of combinations of sequences mimicking the sequences in **antibodies** of different sources, whether these sources are differing classes, differing antigen responses, or differing species of origin, and whether or not the **fusion** point is at the variable/constant boundary. For instance, chimeric **antibodies** can include **antibodies** where the

framework and
complementarity-determining regions are from different sources. For example, non-human CDRs are integrated into human framework regions linked to a human constant region to make **humanized antibodies**. See,, for example,, PCT Application Publication No. WO 87/02671, U.S. Pat. No.

. . .
0173494, Jones, et al.,
Natu.re 321:522-525 (1986) and Verhoeyen, et al., Science 239:1534-1536 (1988)e
A human-like framework region is a framework region for each **antibody** chain, and it usually comprises at least about 70 or more amino acid residues, typically 75 to 85 or more residues. The. . .

The term **humanized** or human-like immunoglobulin refers to an immunoglobulin comprising a human-like framework region and a constant region that is substantially homologous to a human. . .

Hybrid **antibody** refers to an **antibody** wherein each chain is separately homologous with reference to a mammalian **antibody** chain, but the combination represents a novel assembly so that two different antigens are recognized by the **antibody**. In hybrid **antibodies**, one heavy and light chain
pair is homologous to that found in an **antibody** raised against one epitope, while the other heavy and light chain pair is homologous to a pair found in an **antibody** raised against another epitope, This results in the property of multi-functional valency or multivalency, i.e., ability to bind at least two different. . .

The present invention encompasses, inter alia,,, a chimeric **antibody**, including a hybrid **antibody** or a **humanized**
or human-like **antibody**, It also encompasses a recombinant DNA sequence encoding segments of the **antibody** or any peptide specific for vitronectin or a fragment of vitronectin.

For this invention, an immunoglobulin, **antibody** or other peptide is specific for vitronectin or a fragment thereof if the immunoglobulin **antibody** or peptide binds or is capable of binding vitronectin or the fragment as measured or determined by standard **antibody**-antigen or ligand-receptor assays. Examples of such assays include competitive assays,, saturation assays, and standard immunoassays such as ELISA or RIAe This definition of specificity applies to single heavy and/or light chains, CDRs. **fusion** proteins or fragments of heavy and/or light chains, that are also specific for vitronectin if they bind vitronectin alone or if, when properly. . .

In competition assays, the ability of an **antibody** or peptide fragment to bind an antigen such as vitronectin is determined by detecting the ability of the peptide to compete with the. . .

. . .
using a competition assay are
also available. For instance, immunoglobulins can be used to identify the presence of vitronectin, Standard procedures for monoclonal **antibody** assays, such as ELISA, may be used (see, Harlow and Lane, supra). For a review of various signal producing systems which may. . .

To identify **antibodies** with the desired specificity a number of well-defined techniques are known and can be applied to methods of the invention. Such techniques relate to, for example, the **antibodies'** ability to stain tissue or deposits via histochemical means, to react with intact tissue on a Fluorescence-activated cell sorter (FACS), or to. . .

. . .

that are well known in the art, the variable regions and CDRs may be derived from a hybridoma that produces a monoclonal **antibody** that is specific for vitronectin. Nucleic acid sequences relating to the present invention which are capable of ultimately expressing the desired chimeric **antibodies** can be formed from a variety of different nucleotide sequences (genomic or cDNA, RNA, synthetic oligonucleotides, etc.) and components (e.g., VI J, D,. . .

. . .

(1988); Liu, et al., PNAS USA 84:3439 (1987)o The CDRs for producing the immunoglobulins of the present invention preferably are derived from monoclonal **antibodies** capable of binding to the desired antigen, vitronectin receptor protein, and produced in any convenient mammalian source, including, mice, rats, rabbits, hamsters, or other vertebrate host cells capable of producing **antibodies** by well known methods. Suitable source cells for the DNA sequences and host cells for immunoglobulin expression and secretion can be obtained from. . .

In addition to the **antibody** peptides described herein, other substantially homologous modified immunoglobulins can be readily designed and manufactured utilizing various recombinant DNA and synthetic techniques known to those. . . and Smith, Gene 8:81-97 (1979) and Roberts, S., et al., Nature 328:731-734 (1987). Alternatively, polypeptide fragments comprising only a portion of the primary **antibody** structure may be produced, which fragments possess binding and/or effector activities.

For example, the DNA sequence encoding the chimeric **antibody** amino acid sequence can be linked to yeast promoters and enhancers and transfected into yeast by methods well known in the art.

. . .

as a different mammalian species. The CDRs can then be ligated to the framework regions and constant regions to form a chimeric **antibody**. See PCT No. GB88/00731 (1989), The CDRs could be cloned in an expression vector comprising, for example, human framework and constant regions.

. . .

chain CDR1, CDR2, and CDR3 of one species, such as mouse, and the framework regions of human heavy chain to encode an **antibody** specific for vitronectin. Other possibilities include using CDRs specific for vitronectin; using part of the variable region encompassing CDR1 and CDR2 from one. . .

Antibodies may be expressed in an appropriate folded form, including **single chain antibodies**, from bacteria such as E. coli. See Pluckthun, Biotechnology 9:545 (1991); Huse, et al., Science 246:1275 (1989) and Ward, et al., Nature 341:544 (1989) e

For diagnostic purposes, the immunoglobulins may either be labeled or unlabeled, Unlabeled **antibodies** can be used in combination with other labeled **antibodies** (second **antibodies**) that are reactive with the first **antibody**, such as **antibodies** specific for human immunoglobulin constant regions.

Alternatively, the **antibodies** can be directly labeled. A wide variety of labels may be employed, such as radionuclides, fluors including fluorophores and fluorochromes, chromophores, enzymes, enzyme. . .

. . .

compounds may be synthesized with similar biological activity by first determining the relevant contact residues and conformation involved in vitronectin binding by an **antibody** peptide of this invention. Computer programs to create models of proteins such as **antibodies** are generally available and well known to those skilled in the art. See Kabat, et al., Sequences of Proteins of immunological Interest,0,. . . organic molecules can be synthesized. See, for example, Saragovi, et al., Science 253:792 (1991)

Purification of Protein

The invention provides proteins such as anti-vitronectin **antibodies**, Protein purification is known in the art. The proteins of the invention can be purified according to standard procedures of the art,. . .

. . .

region of the deposits could be used as therapeutic or diagnostic agents. Such compounds are examples of vitronectin-ameliorative compounds and can include anti-vitronectin **antibodies**, vitronectin receptor molecules (integrins), thrombin, anti-thrombin-3, thrombospondin, thrombomodulin, heparin,, heparan sulfate, heparin cofactor 2,, plasminogen, tissue plasminogen activator (TPA), plasminogen activator inhibitors, endorphins, amyloid, serum amyloid P component, coumadin, somatomedin B. C5b-9 complement complex, fibrin, keratin, elastin, perforin, factor X, transglutaminase, protein kinases, sulfotransferases, trypsin-like protease, **nidogen**, osteopontin, transforming growth factor-fl (TGF-fl) and other vitronectin-binding molecules or specific amino acid or other molecular sequences derived from such compounds or derived. . .

. . .

tumor diagnosis. See Miettinen, M., Annals of Medicine 25:221-233 (1993). similarly, the art accepts the correlation between structures labeled in vivo by fluorochrome-labeled **antibodies** and structures labeled in histologic sections examined by fluorescence microscopy* See Scheiffarth (1990)o

Preferred compounds for use with the invention are those which act. . . amongst such compounds are those with monovalent binding characteristics and without other functional characteristics that might cause undesirable side effects. For instance, monovalent anti-vitronectin **antibody** fragments (e.g. Fab fragments derived from proteolytic cleavage of IgG or **antibody** fragments obtained by recombinant DNA cloning and expression) and/or relatively inert vitronectin-binding polypeptides derived synthetically or by cleavage of known vitronectin-binding proteins are preferred. . .

For example, monovalent **antibody** fragments directed against one or more vitronectin-binding molecules (e.g. Fab fragments derived from proteolytic cleavage of IgG or **antibody** fragments obtained by recombinant DNA cloning and expression) and/or relatively inert polypeptides (derived synthetically orby proteolytic cleavage) with the capacity to bind one. . .

. . .
by the expression of genetically engineered recombinant proteins. For example, the vitronectin-binding domain(s) of the heavy and/or light chains of an anti-vitronectin **antibody** could be coupled to a proteolytic enzyme known to digest vitronectin. Alternatively, the genetic sequences encoding these two molecular species may combined and. . .

. . .
or practitioner administering the therapy are among the factors affecting the selected dosage. For example, the dosage of an immunoglobulin such as an **antibody** will range from about 1.0 microgram per kilogram per day to about 1 milligram per kg per day for polyclonal **antibodies** and about 5% to about 10% of that amount for monoclonal **antibodies**. In such a case, the immunoglobulin can be administered once daily as an intravenous infusion.

. . .
Vitronectin Probes
The invention provides vitronectin probes or compounds which specifically bind to vitronectin. Usually the probe is a peptide such as an **antibody**.

The use of certain labeled **antibodies** for purposes other than the invention is known. For example, fluorescein-labeled **antibodies** have been injected into the ear vein of a rabbit and visualized in the eye up to 24 hours later. The specific binding of the **antibody** probes to the targeted chorioretinal lesions was confirmed in subsequent histologic examination of the ocular tissue using fluorescence light microscopy. See Scheiffarth at page 275. Similar use of **antibody** probes in humans has been documented for tumor immunodetection and immunotherapy. See, for example, Miettinen (1993).

Intravenously injected **antibodies** or fluorescein (fluorescein angiography is well known in the art) are capable of reaching the retina/choroid region of the eye. Because the endothelial. . . the site at which drusen deposits are formed. Since the extravascular space presents no barrier to the diffusion of proteins such as **antibodies** or other drusen-binding molecules, the intravenously applied anti-drusen probes have free access to their target ligands.

Also, there do not appear to be any adverse, short-term effects to administration of labeled **antibodies** targeting the eye or other compartments, The in vivo use of **antibodies** in humans for diagnostic and therapeutic purposes has demonstrated significant long-term tolerance, particularly with modifications such as **humanized antibodies** or **single chain**, **single** domain or bioengineered **antibody** fragments discussed herein. See also, for example, Maraveyas, A. and A. A. Epenetos, Cancer Immunol. immunother. 34:71-73 (1991) and De Jager et al., Seminars in Nuc. Med. 23(2):165-179 (1993)e Finally, many of the inventive anti-drusen probes, in addition to **antibodies** directed against specific drusen-associated molecules, are normal components of blood plasma and/or extracellular matrix and thus would not produce adverse side effects.

Accordingly, labeled drusen-binding **antibodies** and other drusen-binding molecules can reach and bind to drusen deposits in the eye following intravenous injection or other routes of administration.

The vitronectin probe is not limited to **antibodies**, however. Any agent that binds to vitronectin could be used.

. . .
Additional fixed tissue from each eye was processed for correlative examination by electron microscopy. Individual sections were examined immunohistochemically using a variety of **antibodies** and lectins. Other sections from the same eye(s) served as the controls.

. . .
of the immunolabeling. These controls included sections incubated in solutions in place of, or in addition to, the solutions containing the experimental primary **antibody** (lectins). These control solutions were applied at the same weight to volume concentration as in the experimental condition; they contained one of the following reagents: pre-immune serum, non-immune serum, an irrelevant **antibody** or lectin, primary **antibody** plus an excess of antigen, or buffer solution with or without bovine serum albumin.

. . .
were separated using one-dimensional SDS-polyacrylamide gel electrophoresis (PAGE) and transferred to nitrocellulose paper. The isolated proteins were probed with a panel of **antibodies** and lectins, including those listed in Table III. Drusen-enriched preparations showed numerous labeled bands varying in molecular weight from 5,000 to 300,000 daltons. . .

. . .
the early immunohistochemical evidence of vitronectin deposition associated with drusen. Most striking were small nuclei (less than 1 gm diameter) that reacted with **antibodies** directed against vitronectin, Type IV collagen and wheat germ agglutinin.

. . .
and embedded in acrylamide and sectioned to a thickness of 5-8 Am on a cryostat at -200C, Sections were incubated with various drusen-reactive **antibodies** (including **antibodies** directed against vitronectin) and lectins (see Table III), as well as with hematoxylin and eosin (HE) and periodic acid Schiff (PAS) stains. . .

. . .
removed drusen, Also, treatment with the proteases chymotrypsin, collagenase, dispase, elastase, Factor Xa and trypsin reduced the binding of some drusen-binding lectins and **antibodies**.

. . .
be an immunoassay, such as an enzyme-linked i'mmunoassay (ELISA), which detects in serum one or more of the following: 1) presence of vitronectin **antibodies**; 2) abnormal levels of vitronectin protein; 3) abnormal vitronectin isoform ratios; and 4) aberrant forms of vitronectin. For example, patients with serum. . .

The presence of **antibodies** directed against components of drusen and other deposits known as basal linear deposits has been identified in the sera of some patients. . . 35 patients with AMD was applied to sections of eyes containing drusen and other abnormal deposits, followed by application of a human-specific

secondary **antibody** conjugated with fluorescein. Approximately 30% of the serum samples bound specifically and intensely to drusen and other deposits in Bruch's membrane.

Western blot analyses in which purified human vitronectin (Vn) was exposed to sera from AMD patients demonstrated vitronectin binding **antibodies** in some serum samples. Also, sera from some donors with AMD contained various aberrant electrophoretic bands of 25, 29, 30 and 80 kDa. . .

. . .
or having a significant predisposition to AMD warranting prophylactic intervention is selected for a clinical diagnostic trial. A drusen-binding molecule(s), such as vitronectin **antibody**, vitronectin protein, or vitronectin protein fragment, is conjugated to an appropriate fluorochrome, such as fluorescein, using methods well known in the art. The. . .

The trabecular meshwork-containing tissues were fixed and prepared for immunocytochemical observations. Many of these specimens demonstrated a strong positive reaction with anti-vitronectin **antibodies**. Control specimens, collected from human eyes derived from donors without glaucoma did not exhibit the same reaction. These studies suggest that vitronectin-containing deposits. . .

TABLE I
VITRONECTIN@BINDING MOLECULES*
Alpha-1 Proteinase Inhibitor
Anti-Vitronectin **Antibodies** (and fragments thereof)
Amyloid
Amyloid P Component
Collagens
Coumadin
C5b-9 Complement Complex
Dextran Sulfate
Elastin/Elastic Tissue Fibers
E-Endorphin
Factor X
Factor XIII (Plasma transglutaminase)
Fibrillin
Fibrin
Fucoidan
Growth Factors (e.g. TGF-B)
Heparan Sulfate
Heparin
Heparin Cofactor 2
Integrins (Cell membrane-associated Vn receptors)
Keratin
Nidogen
Osteopontin
Perforin
Plasminogen
Plasminogen Activators
Plasminogen Activator Inhibitor-1 (PAI-1)
Platelet Membrane Glycoprotein IIb-IIIa (GPIIb-IIIa)
Protein kinases
Somatomedin B
Sulfotransferases
Thrombin/Antithrombin III
Thrombomodulin
Thrombospondin
Transforming Growth Factor-B (TGF-B)
Transglutaminase
Trypsin-Like Protease
* See text for preferred dosages. . .

glucosides
Bile Acids is
Lecithins &
Lyso- to
lecithins
Ethanol &
Other
Alcohols
*Preferred **Alternative sources are7a=aila=le
SUBSTITUTE SHEET (RULE 26)
TABLE III
DRUMEN-BINDING PROBES
A) **ANTIBODIES** DIRECTED AGAINST
Vitronectin
Anyloid P Component
Chandroitin Sulfate Proteoglycan
Heparan Sulfate Proteoglycan
Apolipoprotein E
Thrombospondin
Elastin
Complement component Clq
Complement C5-9A complex
Factor X
Plasminogen
a,-Antichymotrypsin
Thrombin
Thrombospondin
Fibrinogen
Plasminogen
Elastin
Cystatin C
Haptoglobin
Prealbumin
Immunoglobulin lambda chain
HLA-DR
B) LECTINS*
RCA: Ricinus co=unis. . .

CLMEN 7 The method of claim 6 wherein the vitronectin-binding molecule is a monovalent anti-vitronectin **antibody**.
a, The method of claim 6 wherein the glycosidase is selected from the group consisting of endoglycosidase-F and chondroitinase.
91 The method of claim. . .

23 The method of claim 22 wherein the vitronectin-binding molecule is a monovalent monoclonal **antibody**,

26 The method of claim 24 wherein the vitronectin probe is a monovalent monoclonal **antibody** raised against vitronectin.

=> d his

(FILE 'HOME' ENTERED AT 13:34:50 ON 24 APR 2003)

FILE 'MEDLINE, CANCERLIT, BIOSIS, CONFSCI, CAPLUS, EMBASE, USPATFULL, PCTFULL, SCISEARCH' ENTERED AT 13:35:24 ON 24 APR 2003
L1 713 S NIDOGEN AND ANTIBOD?
L2 407 DUP REM L1 (306 DUPLICATES REMOVED)
L3 244 S L2 NOT PY=>2000

```
L4            45 S L3 AND FUSION
L5            10 S L4 AND HUMANIZE?
L6             9 S L5 AND SINGLE (A) CHAIN

=> s nidogen (10a) antibod?
L7          142 NIDOGEN (10A) ANTIBOD?

=> s l7 not py=>1999
'1999' NOT A VALID FIELD CODE
L8          101 L7 NOT PY=>1999

=> dup rem l8
PROCESSING COMPLETED FOR L8
L9            29 DUP REM L8 (72 DUPLICATES REMOVED)

=> d 1-29

L9      ANSWER 1 OF 29     PCTFULL   COPYRIGHT 2003 Univentio
AN      1998031709 PCTFULL  ED 20020514
TIEN    ANTIBODIES THAT BIND TO THE NIDOGEN-BINDING DOMAIN
        OF LAMININ, THEIR PRODUCTION AND USE
TIFR    ANTICORPS QUI SE LIENT AUX DOMAINES DE LIAISON DE NIDOGENE DE LA
        LAMININE, LEUR PRODUCTION ET LEUR UTILISATION
IN      GERL, Martin
PA      HOECHST AKTIENGESELLSCHAFT;
        GERL, Martin
LA      German
DT      Patent
PI      WO 9831709            A1 19980723
DS      W:          AU BR CA CN CZ HU ID IL JP KR MX PL RU TR US AT BE CH DE DK
                    ES FI FR GB GR IE IT LU MC NL PT SE
AI      WO 1997-EP7241        A  19971222
PRAI    DE 1997-197 01 607.3     19970117
ICM     C07K016-18
ICS     C07K016-46; C12N005-20; A61K039-00; A61K039-395; G01N033-68;
        G01N033-577

L9    ANSWER 2 OF 29     CANCERLIT                          DUPLICATE 1
AN    1998311650     CANCERLIT
DN    98311650    PubMed ID: 9647658
TI    The laminin-nidogen complex is a ligand for a specific splice isoform of
      the transmembrane protein tyrosine phosphatase LAR.
AU    O'Grady P; Thai T C; Saito H
CS    Dana-Farber Cancer Institute and Department of Biological Chemistry and
      Molecular Pharmacology, Harvard Medical School, Boston, Massachusetts
      02115, USA.
NC    GM53415 (NIGMS)
SO    JOURNAL OF CELL BIOLOGY, (1998 Jun 29) 141 (7) 1675-84.
      Journal code: 0375356. ISSN: 0021-9525.
CY    United States
DT    Journal; Article; (JOURNAL ARTICLE)
LA    English
FS    MEDLINE; Priority Journals
OS    MEDLINE 1998311650
EM    199808
ED    Entered STN: 19980910
      Last Updated on STN: 19980910

L9    ANSWER 3 OF 29     MEDLINE                            DUPLICATE 2
AN    1998406162     MEDLINE
DN    98406162    PubMed ID: 9733643
TI    Nidogen-2: a new basement membrane protein with diverse binding
      properties.
AU    Kohfeldt E; Sasaki T; Gohring W; Timpl R
CS    Max-Planck-Institut fur Biochemie, D-82152 Martinsried, Germany.
SO    JOURNAL OF MOLECULAR BIOLOGY, (1998 Sep 11) 282 (1) 99-109.
      Journal code: 2985088R. ISSN: 0022-2836.
CY    ENGLAND: United Kingdom
DT    Journal; Article; (JOURNAL ARTICLE)
LA    English
FS    Priority Journals
OS    GENBANK-AJ223500
EM    199810
ED    Entered STN: 19981021
      Last Updated on STN: 19981021
      Entered Medline: 19981015

L9    ANSWER 4 OF 29     MEDLINE                            DUPLICATE 3
AN    97195710     MEDLINE
DN    97195710    PubMed ID: 9043083
TI    Importance of nidogen binding to laminin gamma1 for branching epithelial
      morphogenesis of the submandibular gland.
AU    Kadoya Y; Salmivirta K; Talts J F; Kadoya K; Mayer U; Timpl R; Ekblom P
CS    Department of Animal Physiology, Uppsala University, Biomedical
      Center,Sweden.
SO    DEVELOPMENT, (1997 Feb) 124 (3) 683-91.
      Journal code: 8701744. ISSN: 0950-1991.
CY    ENGLAND: United Kingdom
DT    Journal; Article; (JOURNAL ARTICLE)
LA    English
FS    Priority Journals
EM    199703
ED    Entered STN: 19970407
      Last Updated on STN: 20000303
      Entered Medline: 19970325

L9    ANSWER 5 OF 29  USPATFULL
AN      96:14906  USPATFULL
TI      Two non-contiguous regions contribute to nidogen binding to a single
        EGF-like motif of the laminin .gamma.1 chain
IN      Fox, Jay W., Charlottesville, VA, United States
        Timpl, Rupert, Martinsried, Germany, Federal Republic of
PA      The University Of Virginia Patent Foundation, Charlottesville, VA,
        United States (U.S. corporation)
PI      US 5493008              19960220
AI      US 1994-288728          19940815 (8)
DT      Utility
FS      Granted
LN.CNT 981
INCL    INCLM: 530/326.000
        INCLS: 530/324.000; 530/327.000; 530/328.000; 530/329.000
NCL     NCLM:  530/326.000
        NCLS:  530/324.000; 530/327.000; 530/328.000; 530/329.000
IC      [6]
        ICM: A61K038-00
        ICS: C07K005-00
EXF     530/324; 530/326; 530/327; 530/328; 530/329
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9      ANSWER 6 OF 29     PCTFULL   COPYRIGHT 2003 Univentio
AN      1996004926 PCTFULL  ED 20020514
TIEN    TWO NON-CONTIGUOUS REGIONS CONTRIBUTE TO NIDOGEN BINDING TO A SINGLE
        EGF-LIKE MOTIF OF THE LAMININ 'gamma'1 CHAIN
TIFR    DEUX REGIONS NON CONTIGUES CONTRIBUANT A LA LIAISON NIDOGENE AVEC UN
        MOTIF UNIQUE DU TYPE EGF DE LA CHAINE 'gamma'1 DE LA LAMININE
IN      FOX, Jay, W.;
        TIMPL, Rupert
PA      THE UNIVERSITY OF VIRGINIA PATENT FOUNDATION
LA      English
DT      Patent
```

PI WO 9604926 A1 19960222
DS W: AU CA JP AT BE CH DE DK ES FR GB GR IE IT LU MC NL PT SE
AI WO 1995-US9693 A 19950811
PRAI US 1994-288,728 19940815
ICM A61K038-00
ICS A61K038-04

L9 ANSWER 7 OF 29 MEDLINE DUPLICATE 4
AN 96007609 MEDLINE
DN 96007609 PubMed ID: 7561165
TI Skin fibroblasts are the only source of nidogen during early basal lamina formation in vitro.
AU Fleischmajer R; Schechter A; Bruns M; Perlish J S; Macdonald E D; Pan T C; Timpl R; Chu M L
CS Department of Dermatology, Mount Sinai School of Medicine, New York, New York 10029, USA.
SO JOURNAL OF INVESTIGATIVE DERMATOLOGY, (1995 Oct) 105 (4) 597-601.
Journal code: 0426720. ISSN: 0022-202X.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199511
ED Entered STN: 19951227
Last Updated on STN: 20000303
Entered Medline: 19951114

L9 ANSWER 8 OF 29 MEDLINE DUPLICATE 5
AN 95009530 MEDLINE
DN 95009530 PubMed ID: 7925005
TI Role of mesenchymal nidogen for epithelial morphogenesis in vitro.
AU Ekblom P; Ekblom M; Fecker L; Klein G; Zhang H Y; Kadoya Y; Chu M L; Mayer U; Timpl R
CS Department of Animal Physiology, Uppsala University, Sweden.
NC AR 38923 (NIAMS)
SO DEVELOPMENT, (1994 Jul) 120 (7) 2003-14.
Journal code: 8701744. ISSN: 0950-1991.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199411
ED Entered STN: 19941222
Last Updated on STN: 19941222
Entered Medline: 19941101

L9 ANSWER 9 OF 29 MEDLINE DUPLICATE 6
AN 95051016 MEDLINE
DN 95051016 PubMed ID: 7962110
TI Influence of nidogen complexed or not with laminin on attachment, spreading, and albumin and laminin B2 mRNA levels of rat hepatocytes.
AU Levavasseur F; Mayer U; Guillouzo A; Clement B
CS Unite de Recherches Hepatologiques, INSERM U-49, Hopital Pontchaillou, Rennes, France.
SO JOURNAL OF CELLULAR PHYSIOLOGY, (1994 Nov) 161 (2) 257-66.
Journal code: 0050222. ISSN: 0021-9541.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199412
ED Entered STN: 19950110
Last Updated on STN: 19950110
Entered Medline: 19941228

L9 ANSWER 10 OF 29 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.DUPLICATE 7
AN 1993:342697 BIOSIS
DN PREV199396039697
TI A single EGF-like motif of laminin is responsible for high affinity nidogen binding.
AU Mayer, Ulrike; Nischt, Roswitha; Poeschl, Ernst; Mann, Karlheinz; Fukuda, Katsunori; Gerl, Martin; Yamada, Yoshihiko; Timpl, Rupert (1)
CS (1) Max-Planck-Inst. Biochem., D-8033 Martinsried Germany
SO EMBO (European Molecular Biology Organization) Journal, (1993) Vol. 12, No. 5, pp. 1879-1885.
ISSN: 0261-4189.
DT Article
LA English

L9 ANSWER 11 OF 29 MEDLINE DUPLICATE 8
AN 93146648 MEDLINE
DN 93146648 PubMed ID: 8425764
TI Myoepithelial and basement membrane antigens in benign and malignant human breast tumors.
AU Guelstein V I; Tchypysheva T A; Ermilova V D; Ljubimov A V
CS Cancer Research Center, Russian Academy of Medical Sciences, Moscow.
SO INTERNATIONAL JOURNAL OF CANCER, (1993 Jan 21) 53 (2) 269-77.
Journal code: 0042124. ISSN: 0020-7136.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199303
ED Entered STN: 19930312
Last Updated on STN: 19930312
Entered Medline: 19930304

L9 ANSWER 12 OF 29 MEDLINE DUPLICATE 9
AN 93238676 MEDLINE
DN 93238676 PubMed ID: 8477687
TI Ascidian entactin/nidogen. Implication of evolution by shuffling two kinds of cysteine-rich motifs.
AU Nakae H; Sugano M; Ishimori Y; Endo T; Obinata T
CS Advanced Research Laboratory, Research and Development Center, Toshiba Corporation, Japan.
SO EUROPEAN JOURNAL OF BIOCHEMISTRY, (1993 Apr 1) 213 (1) 11-9.
Journal code: 0107600. ISSN: 0014-2956.
CY GERMANY: Germany, Federal Republic of
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-D14038; GENBANK-L09679; GENBANK-L09680; GENBANK-L09681; GENBANK-L09682; GENBANK-L09683; GENBANK-X57950; GENBANK-X70793; GENBANK-X70999; GENBANK-X71000
EM 199305
ED Entered STN: 19930611
Last Updated on STN: 20000303
Entered Medline: 19930521

L9 ANSWER 13 OF 29 MEDLINE DUPLICATE 10
AN 92165419 MEDLINE
DN 92165419 PubMed ID: 1371500
TI Distribution of individual components of basement membrane in human colon polyps and adenocarcinomas as revealed by monoclonal antibodies.
AU Ljubimov A V; Bartek J; Couchman J R; Kapuller L L; Veselov V V; Kovarik J; Perevoshchikov A G; Krutovskikh V A
CS All-Union Cancer Research Center, USSR AMS, Moscow.
NC AR36457 (NIAMS)
SO INTERNATIONAL JOURNAL OF CANCER, (1992 Feb 20) 50 (4) 562-6.

Journal code: 0042124. ISSN: 0020-7136.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199203
ED Entered STN: 19920417
Last Updated on STN: 19980206
Entered Medline: 19920330

L9 ANSWER 14 OF 29 MEDLINE DUPLICATE 11
AN 94218359 MEDLINE
DN 94218359 PubMed ID: 1344818
TI Patterns of basement membrane laminin distribution in nonneoplastic and neoplastic thyroid tissue.
AU Campo E; Perez M; Charonis A A; Axiotis C A; Merino M J
CS Laboratory of Pathology, National Institutes of Health, Bethesda, Maryland.
SO MODERN PATHOLOGY, (1992 Sep) 5 (5) 540-6.
Journal code: 8806605. ISSN: 0893-3952.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199405
ED Entered STN: 19940606
Last Updated on STN: 19940606
Entered Medline: 19940524

L9 ANSWER 15 OF 29 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.DUPLICATE 12
AN 1993:95611 BIOSIS
DN PREV199395050807
TI Characterization of a natural human antibody with anti-galactosyl(alpha-1-2)galactose specificity that is present at high titers in chronic Trypanosoma cruzi infection.
AU Avila, Jose Luis; Rojas, Miguel; Velaquez-Avila, Gladys
CS Inst. Biomed. Caracas, Venezuela, Hosp. de Ninos J. M. de los Rios, Caracas Venezuela
SO American Journal of Tropical Medicine and Hygiene, (1992) Vol. 47, No. 4, pp. 413-421.
ISSN: 0002-9637.
DT Article
LA English

L9 ANSWER 16 OF 29 MEDLINE DUPLICATE 13
AN 92111677 MEDLINE
DN 92111677 PubMed ID: 1370418
TI American Leishmania spp. and Trypanosoma cruzi: galactosyl alpha(1-3) galactose epitope localization by colloidal gold immunocytochemistry and lectin cytochemistry.
AU Bretana A; Avila J L; Contreras-Bretana M; Tapia F J
CS Secci+SUon de Microscopia Electronica, Instituto de Biomedicina, Caracas, Venezuela.
SO EXPERIMENTAL PARASITOLOGY, (1992 Feb) 74 (1) 27-37.
Journal code: 0370713. ISSN: 0014-4894.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199202
ED Entered STN: 19920308
Last Updated on STN: 19960129
Entered Medline: 19920218

L9 ANSWER 17 OF 29 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.DUPLICATE 14
AN 1990:518268 BIOSIS
DN BA90:135544
TI ULTRASTRUCTURAL LOCALIZATION OF THE CORE PROTEIN OF A BASEMENT MEMBRANE-SPECIFIC CHONDROITIN SULFATE PROTEOGLYCAN IN ADULT RAT SKIN.
AU MCCARTHY K J; HORIGUCHI Y; COUCHMAN J R; FINE J-D
CS DEP. CELL BIOL. AND ANAT., VH 201 C BOX 803, UNIV. ALA. BIRMINGHAM, BIRMINGHAM, ALA. 35294, USA.
SO ARCH DERMATOL RES, (1990) 282 (6), 397-401.
CODEN: ADREDL. ISSN: 0340-3696.
FS BA; OLD
LA English

L9 ANSWER 18 OF 29 MEDLINE DUPLICATE 15
AN 90384093 MEDLINE
DN 90384093 PubMed ID: 2119467
TI Entactin: a possible auto-antigen in the pathogenesis of non-Goodpasture anti-GBM nephritis.
AU Saxena R; Bygren P; Butkowski R; Wieslander J
CS Department of Nephrology, University Hospital of Lund, Sweden.
SO KIDNEY INTERNATIONAL, (1990 Aug) 38 (2) 263-72.
Journal code: 0323470. ISSN: 0085-2538.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199010
ED Entered STN: 19901122
Last Updated on STN: 19980206
Entered Medline: 19901026

L9 ANSWER 19 OF 29 MEDLINE DUPLICATE 16
AN 90118740 MEDLINE
DN 90118740 PubMed ID: 2481931
TI An improved immunofluorescence technique for the histological examination of blood vessel tissue.
AU Kittelberger R; Davis P F; Stehbens W E
CS Malaghan Institute of Medical Research, Wellington School of Medicine, Wellington Hospital, New Zealand.
SO ACTA HISTOCHEMICA, (1989) 86 (2) 137-42.
Journal code: 0370320. ISSN: 0065-1281.
CY GERMANY, EAST: German Democratic Republic
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199002
ED Entered STN: 19900328
Last Updated on STN: 19960129
Entered Medline: 19900220

L9 ANSWER 20 OF 29 MEDLINE
AN 90143096 MEDLINE
DN 90143096 PubMed ID: 2482635
TI [Structure and antigenicity of the glomerular basement membrane]. Aufbau und Antigenitat der glomerularen Basalmembran.
AU Weber M
SO VERHANDLUNGEN DER DEUTSCHEN GESELLSCHAFT FUR PATHOLOGIE, (1989) 73 6-12.
Ref: 38
Journal code: 7503704. ISSN: 0070-4113.
CY GERMANY, WEST: Germany, Federal Republic of
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA German

FS Priority Journals
EM 199003
ED Entered STN: 19900328
Last Updated on STN: 19960129
Entered Medline: 19900312

L9 ANSWER 21 OF 29 CAPLUS COPYRIGHT 2003 ACS
AN 1988:567788 CAPLUS
DN 109:167788
TI High resolution immunoelectron microscopic localization of functional domains of laminin, nidogen, and heparan sulfate proteoglycan in epithelial basement membrane of mouse cornea reveals different topological orientations
AU Schittny, Johannes C.; Timpl, Rupert; Engel, Juergen
CS Biocent., Univ. Basel, Basel, CH-4056, Switz.
SO Journal of Cell Biology (1988), 107(4), 1599-610
CODEN: JCLBA3; ISSN: 0021-9525
DT Journal
LA English

L9 ANSWER 22 OF 29 MEDLINE DUPLICATE 17
AN 88151991 MEDLINE
DN 88151991 PubMed ID: 3126070
TI Analysis of degradation of the basement membrane protein **nidogen**, using a specific monoclonal **antibody**.
AU Dziadek M; Clements R; Mitrangas K; Reiter H; Fowler K
CS Murdoch Institute for Research into Birth Defects, Royal Children's Hospital, Parkville, Victoria, Australia.
SO EUROPEAN JOURNAL OF BIOCHEMISTRY, (1988 Feb 15) 172 (1) 219-25.
Journal code: 0107600. ISSN: 0014-2956.
CY GERMANY, WEST: Germany, Federal Republic of
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198804
ED Entered STN: 19900308
Last Updated on STN: 19900308
Entered Medline: 19880419

L9 ANSWER 23 OF 29 SCISEARCH COPYRIGHT 2003 ISI (R)
AN 88:109325 SCISEARCH
GA The Genuine Article (R) Number: M2364
TI ANALYSIS OF DEGRADATION OF THE BASEMENT-MEMBRANE PROTEIN **NIDOGEN**, USING A SPECIFIC MONOCLONAL-**ANTIBODY**
AU DZIADEK M (Reprint); CLEMENTS R; MITRANGAS K; REITER H; FOWLER K
CS ROYAL CHILDRENS HOSP, MURDOCH INST RES BIRTH DEFECTS, PARKVILLE, VIC 3052, AUSTRALIA
CYA AUSTRALIA
SO EUROPEAN JOURNAL OF BIOCHEMISTRY, (1988) Vol. 172, No. 1, pp. 219-225.
DT Article; Journal
FS LIFE
LA ENGLISH
REC Reference Count: 42

L9 ANSWER 24 OF 29 MEDLINE DUPLICATE 18
AN 88139674 MEDLINE
DN 88139674 PubMed ID: 2449451
TI Serological activity against galactosyl-alpha(1-3)galactose in sera from patients with several kinetoplastida infections.
AU Avila J L; Rojas M; Towbin H
CS Instituto de Biomedicina, Caracas, Venezuela.
SO JOURNAL OF CLINICAL MICROBIOLOGY, (1988 Jan) 26 (1) 126-32.
Journal code: 7505564. ISSN: 0095-1137.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198804
ED Entered STN: 19900308
Last Updated on STN: 19900308
Entered Medline: 19880407

L9 ANSWER 25 OF 29 MEDLINE DUPLICATE 19
AN 87308118 MEDLINE
DN 87308118 PubMed ID: 3114248
TI The cellular interactions of laminin fragments. Cell adhesion correlates with two fragment-specific high affinity binding sites.
AU Aumailley M; Nurcombe V; Edgar D; Paulsson M; Timpl R
SO JOURNAL OF BIOLOGICAL CHEMISTRY, (1987 Aug 25) 262 (24) 11532-8.
Journal code: 2985121R. ISSN: 0021-9258.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198709
ED Entered STN: 19900305
Last Updated on STN: 19970203
Entered Medline: 19870930

L9 ANSWER 26 OF 29 MEDLINE DUPLICATE 20
AN 88136304 MEDLINE
DN 88136304 PubMed ID: 2449305
TI **Antibodies** to basement membrane proteins **nidogen** and laminin in sera from streptococcal-related diseases and juvenile rheumatoid arthritis patients.
AU Avila J L; Rojas M; Velazquez-Avila G; Rieber M
CS Instituto de Biomedicina, Caracas, Venezuela.
SO CLINICAL AND EXPERIMENTAL IMMUNOLOGY, (1987 Dec) 70 (3) 555-61.
Journal code: 0057202. ISSN: 0009-9104.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198803
ED Entered STN: 19900308
Last Updated on STN: 19900308
Entered Medline: 19880328

L9 ANSWER 27 OF 29 MEDLINE DUPLICATE 21
AN 87034242 MEDLINE
DN 87034242 PubMed ID: 2429987
TI **Antibodies** to basement membrane protein **nidogen** in Chagas' disease and American cutaneous leishmaniasis.
AU Avila J L; Rojas M; Velazquez-Avila G; von der Mark H; Timpl R
SO JOURNAL OF CLINICAL MICROBIOLOGY, (1986 Nov) 24 (5) 775-8.
Journal code: 7505564. ISSN: 0095-1137.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198612
ED Entered STN: 19900302
Last Updated on STN: 19900302
Entered Medline: 19861216

L9 ANSWER 28 OF 29 MEDLINE DUPLICATE 22
AN 86005830 MEDLINE
DN 86005830 PubMed ID: 2995165
TI Expression of nidogen and laminin in basement membranes during mouse embryogenesis and in teratocarcinoma cells.

AU Dziadek M; Timpl R
SO DEVELOPMENTAL BIOLOGY, (1985 Oct) 111 (2) 372-82.
Journal code: 0372762. ISSN: 0012-1606.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198510
ED Entered STN: 19900321
Last Updated on STN: 19900321
Entered Medline: 19851029

L9 ANSWER 29 OF 29 MEDLINE DUPLICATE 23
AN 84108344 MEDLINE
DN 84108344 PubMed ID: 6420150
TI Nidogen: a new, self-aggregating basement membrane protein.
AU Timpl R; Dziadek M; Fujiwara S; Nowack H; Wick G
SO EUROPEAN JOURNAL OF BIOCHEMISTRY, (1983 Dec 15) 137 (3) 455-65.
Journal code: 0107600. ISSN: 0014-2956.
CY GERMANY, WEST: Germany, Federal Republic of
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198403
ED Entered STN: 19900319
Last Updated on STN: 19900319
Entered Medline: 19840301

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L9 ANSWER 1 OF 29 PCTFULL COPYRIGHT 2003 Univentio
ACCESSION NUMBER: 1998031709 PCTFULL ED 20020514
TITLE (ENGLISH): **ANTIBODIES** THAT BIND TO THE **NIDOGEN**
-BINDING DOMAIN OF LAMININ, THEIR PRODUCTION AND USE
TITLE (FRENCH): ANTICORPS QUI SE LIENT AUX DOMAINES DE LIAISON DE NIDOGENE DE LA LAMININE, LEUR PRODUCTION ET LEUR UTILISATION
INVENTOR(S): GERL, Martin
PATENT ASSIGNEE(S): HOECHST AKTIENGESELLSCHAFT;
GERL, Martin
LANGUAGE OF PUBL.: German
DOCUMENT TYPE: Patent
PATENT INFORMATION:

NUMBER	KIND	DATE
WO 9831709	A1	19980723

DESIGNATED STATES
W: AU BR CA CN CZ HU ID IL JP KR MX PL RU TR US AT BE CH DE DK ES FI FR GB GR IE IT LU MC NL PT SE
APPLICATION INFO.: WO 1997-EP7241 A 19971222
PRIORITY INFO.: DE 1997-197 01 607.3 19970117
ABEN Monoclonal and polyclonal antibodies are disclosed as well as parts thereof which bind
specifically to the nidogen-binding domain of laminin, as well as a process for producing the same
and their use as medicaments, as diagnostic agents for detecting laminin isoforms and as model
substances for developing and evaluating substances that influence the **nidogen**-laminin interaction.
The disclosed **antibodies** or their parts bind preferably to the 'gamma'1 III 4 domain of laminin, in
particular in the highly preserved area of loops a and c, and can inhibit the association of laminin
and nidogen.

ABFR L'invention concerne des anticorps monoclonaux et polyclonaux et leurs parties qui se lient
specifiquement au domaine de liaison de nidogene de la laminine, leur procede de production et leur
utilisation comme medicaments, comme agents de diagnostic permettant de detecter des isoformes de la
laminine et comme substances modeles permettant de developper et d'evaluer des substances qui
affectent l'interaction entre le nidogene et la laminine. Ces anticorps ou leurs parties se lient de
preference au domaine 'gamma'1 III 4 de la laminine, surtout dans le domaine tres conserve des
boucles a et c, et peuvent inhiber l'association de la laminine au nidogene.

L9 ANSWER 2 OF 29 CANCERLIT DUPLICATE 1
ACCESSION NUMBER: 1998311650 CANCERLIT
DOCUMENT NUMBER: 98311650 PubMed ID: 9647658
TITLE: The laminin-nidogen complex is a ligand for a specific splice isoform of the transmembrane protein tyrosine phosphatase LAR.
AUTHOR: O'Grady P; Thai T C; Saito H
CORPORATE SOURCE: Dana-Farber Cancer Institute and Department of Biological Chemistry and Molecular Pharmacology, Harvard Medical School, Boston, Massachusetts 02115, USA.
CONTRACT NUMBER: GM53415 (NIGMS)
SOURCE: JOURNAL OF CELL BIOLOGY, (1998 Jun 29) 141 (7) 1675-84.
Journal code: 0375356. ISSN: 0021-9525.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: MEDLINE; Priority Journals
OTHER SOURCE: MEDLINE 1998311650
ENTRY MONTH: 199808
ENTRY DATE: Entered STN: 19980910
Last Updated on STN: 19980910
AB Leukocyte antigen-related protein (LAR) is a prototype for a family of transmembrane protein tyrosine phosphatases whose extracellular domain is composed of three Ig and several fibronectin type III (FnIII) domains. Complex alternative splicing of the LAR-FnIII domains 4-8 has been observed. The extracellular matrix laminin-nidogen complex was identified as a ligand for the LAR-FnIII domain 5 (Fn5) using a series of GST-LAR-FnIII domain fusion proteins and testing them in in vitro ligand-binding assays. LAR- laminin-nidogen binding was regulated by alternative splicing of a small exon within the LAR-Fn5 so that inclusion of this exon sequence resulted in disruption of the laminin-nidogen-binding activity. Long cellular processes were observed when HeLa cells were plated on laminin-**nidogen**, but not when plated on a fibronectin surface. Indirect immunofluorescent **antibody** staining revealed high expression of LAR in a punctate pattern, throughout the length of these cellular processes observed on laminin-**nidogen**. **Antibody**-induced cross-linking of LAR inhibited formation of these cellular processes, and inhibition was correlated with changes in cellular actin cytoskeletal structure. Thus, LAR-laminin-nidogen binding may play a role in regulating cell signaling induced by laminin-nidogen, resulting in cell morphological changes.

L9 ANSWER 3 OF 29 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 1998406162 MEDLINE
DOCUMENT NUMBER: 98406162 PubMed ID: 9733643
TITLE: Nidogen-2: a new basement membrane protein with diverse binding properties.
AUTHOR: Kohfeldt E; Sasaki T; Gohring W; Timpl R
CORPORATE SOURCE: Max-Planck-Institut fur Biochemie, D-82152 Martinsried, Germany.

SOURCE: JOURNAL OF MOLECULAR BIOLOGY, (1998 Sep 11) 282 (1) 99-109.
Journal code: 2985088R. ISSN: 0022-2836.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-AJ223500
ENTRY MONTH: 199810
ENTRY DATE: Entered STN: 19981021
Last Updated on STN: 19981021
Entered Medline: 19981015

AB Human nidogen-2 was cloned and sequenced (1375 residues) and found to share 46% sequence identity and a similar domain arrangement with the previously characterized basement membrane protein nidogen-1. Recombinant nidogen-2 was purified as a 200 kDa protein from transfected mammalian cell medium, showed a high level of N and O-glycosylation, and could be clearly distinguished from **nidogen**-1 (150 kDa) by specific **antibodies**. Electron microscopy demonstrated that the two isoforms have a similar shape, consisting of three globular domains connected by two threads, but differ somewhat in length. Northern blots and immunological assays demonstrated co-expression of the nidogens in various tissues and cultured cells. Immunofluoresence revealed colocalization in vessel walls and other basement membrane zones but some differences in heart and skeletal muscle. Nidogen-2 interacted with collagens I and IV, and perlecan at a comparable level to nidogen-1 but failed to bind to fibulins. Nidogen-2 bound to laminin-1, but only moderately to the epitope on the laminin gammal chain, which promotes high-affinity binding of nidogen-1. Both nidogens were cell-adhesive for a restricted number of cell lines, with nidogen-2 having a higher activity. Together, these data suggest that nidogen-2 can compensate for some but not all functional activities ascribed to nidogen-1.

L9 ANSWER 4 OF 29 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 97195710 MEDLINE
DOCUMENT NUMBER: 97195710 PubMed ID: 9043083
TITLE: Importance of nidogen binding to laminin gammal for branching epithelial morphogenesis of the submandibular gland.
AUTHOR: Kadoya Y; Salmivirta K; Talts J F; Kadoya K; Mayer U; Timpl R; Ekblom P
CORPORATE SOURCE: Department of Animal Physiology, Uppsala University, Biomedical Center,Sweden.
SOURCE: DEVELOPMENT, (1997 Feb) 124 (3) 683-91.
Journal code: 8701744. ISSN: 0950-1991.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199703
ENTRY DATE: Entered STN: 19970407
Last Updated on STN: 20000303
Entered Medline: 19970325

AB Epithelial-mesenchymal interactions are major driving forces for the development of most solid organs. The importance of these interactions was first shown for the embryonic submandibular gland more than 40 years ago. We here present evidence that interactions between two basement membrane components, nidogen (entactin) and laminin gammal chain, could be important for epithelial-mesenchymal interactions in this gland. Nidogen mRNA was detected by in situ hybridization in the mesenchyme, and yet the protein was detected in epithelial and endothelial basement membranes. The role of **nidogen**-laminin interactions for epithelial morphogenesis was studied by applying **antibodies** to submandibular gland organ cultures. **Antibodies** reacting strongly with the **nidogen**-binding site of laminin gammal chain drastically perturbed branching epithelial morphogenesis. Electron microscopy of the epithelial-mesenchymal interface showed that blocking antibodies disrupted the formation of the basement membrane. Epidermal growth factor was shown to increase the expression of **nidogen** in mesenchyme, and could counteract the effect of the blocking **antibodies**. We suggest that nidogen could be an important mesenchymal factor for submandibular gland development.

L9 ANSWER 5 OF 29 USPATFULL
ACCESSION NUMBER: 96:14906 USPATFULL
TITLE: Two non-contiguous regions contribute to nidogen binding to a single EGF-like motif of the laminin .gamma.1 chain
INVENTOR(S): Fox, Jay W., Charlottesville, VA, United States
Timpl, Rupert, Martinsried, Germany, Federal Republic of
PATENT ASSIGNEE(S): The University Of Virginia Patent Foundation, Charlottesville, VA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5493008		19960220
APPLICATION INFO.:	US 1994-288728		19940815 (8)
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	Granted		
PRIMARY EXAMINER:	Warden, Jill		
ASSISTANT EXAMINER:	Prickril, Benet		
LEGAL REPRESENTATIVE:	Oblon, Spivak, McClelland, Maier & Neustadt		
NUMBER OF CLAIMS:	12		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	3 Drawing Figure(s); 2 Drawing Page(s)		
LINE COUNT:	981		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB High affinity binding of nidogen to laminin is mediated by an EGF-like repeat .gamma.1III4 of the mouse laminin .gamma.1 chain and has now been restricted to two short non-contiguous regions of its 56 residue sequence by use of synthetic peptides and recombinant mutants. Disulfide loop a,b of the repeat and a modified loop a,c could completely inhibit binding, with a 5,000-fold or 300-fold reduced affinity, respectively. Synthetic loops c and d lacked inhibitory activity. Some binding contribution of Try819 in loop c was, however, shown by mutation and side chain modification. Together with studies of loop chimeras, this indicated a distinct cooperativity between the two binding sites. The major binding site of loop a was localized to the heptapeptide NIDPNAV (position 798-804). A change of Asp800 to Asn or Ala803 to Val caused a strong reduction in binding activity, while only small effects were observed for the changes Pro801 to Gln and Ile799 to Val. The latter replacement corresponds to the single substitution found in the same region of the Drosophila laminin .gamma.1 chain. However, the changes Asn802 to Ser or Val804 to Ser, both known to exist in the laminin .gamma.2 chain, were deleterious mutations. This demonstrated conservation of binding structure in laminins of distantly related species, but not between homologous chains of laminin isoforms.

L9 ANSWER 6 OF 29 PCTFULL
ACCESSION NUMBER: 1996004926 PCTFULL ED 20020514
TITLE (ENGLISH): TWO NON-CONTIGUOUS REGIONS CONTRIBUTE TO NIDOGEN BINDING TO A SINGLE EGF-LIKE MOTIF OF THE LAMININ 'gamma'1 CHAIN
TITLE (FRENCH): DEUX REGIONS NON CONTIGUES CONTRIBUANT A LA LIAISON NIDOGENE AVEC UN MOTIF UNIQUE DU TYPE EGF DE LA CHAINE 'gamma'1 DE LA LAMININE
INVENTOR(S): FOX, Jay, W.;
TIMPL, Rupert
PATENT ASSIGNEE(S): THE UNIVERSITY OF VIRGINIA PATENT FOUNDATION

LANGUAGE OF PUBL.: English
DOCUMENT TYPE: Patent
PATENT INFORMATION:

NUMBER	KIND	DATE
WO 9604926	A1	19960222

DESIGNATED STATES
W: AU CA JP AT BE CH DE DK ES FR GB GR IE IT LU MC NL PT SE
APPLICATION INFO.: WO 1995-US9693 A 19950811
PRIORITY INFO.: US 1994-288,728 19940815

ABEN The present invention relates to peptide antagonists which specifically prevent laminin interaction with nidogen. Laminin is a major cell-adhesive and structural protein of basement membranes and other extracellular structures occurring as various isoforms of 600-900 kDa, and contains a single high affinity binding site for the 150 kDa basement membrane protein nidogen. The peptide antagonists of this invention may be applied to in vitro studies of organ development or as therapeutic agents for clinical use.

ABFR Cette invention concerne des antagonistes de peptides qui empechent de maniere specifique l'interaction de la laminine avec le nidogene. La laminine est une proteine majeure de structure et d'adhesion cellulaire des membranes basales et d'autres structures extracellulaires se presentant sous diverses isoformes de 600-900 kDa, et contient un seul et unique site de liaison a forte affinite pour le nidogene de proteine de membrane basale a 150 kDa. On peut utiliser les antagonistes de peptides de cette invention dans le cadre des recherches in vitro sur la croissance d'organe ou comme agents therapeutiques destines a un usage clinique.

L9 ANSWER 7 OF 29 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 96007609 MEDLINE
DOCUMENT NUMBER: 96007609 PubMed ID: 7561165
TITLE: Skin fibroblasts are the only source of nidogen during early basal lamina formation in vitro.
AUTHOR: Fleischmajer R; Schechter A; Bruns M; Perlish J S; Macdonald E D; Pan T C; Timpl R; Chu M L
CORPORATE SOURCE: Department of Dermatology, Mount Sinai School of Medicine, New York, New York 10029, USA.
SOURCE: JOURNAL OF INVESTIGATIVE DERMATOLOGY, (1995 Oct) 105 (4) 597-601.
Journal code: 0426720. ISSN: 0022-202X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199511
ENTRY DATE: Entered STN: 19951227
Last Updated on STN: 20000303
Entered Medline: 19951114

AB The purpose of this study was to determine whether nidogen, the linkage protein of the basal lamina, is of epidermal or dermal origin. The development of the basal lamina was studied in an in vitro skin model. Preputial fibroblasts seeded onto a nylon mesh attached, proliferated, and developed a rich extracellular matrix (dermal model). Preputial keratinocytes were added to the dermal model to form a keratinocyte dermal model that ultrastructurally resembled in many respects human skin. Ultrastructural analysis revealed early stages of dermal development, including an incomplete basal lamina, aggregates of dermal filamentous material connecting to the lamina densa, bundles of 10-nm microfibrils, formation of premature hemidesmosomes, anchoring filaments, and anchoring fibrils. The cell origin of nidogen was determined in the dermal model and in the epidermal and dermal components of the keratinocyte dermal model. Specific **antibodies** and a cDNA probe for **nidogen** were used for immunofluorescence microscopy, Western and Northern blots, and for in situ hybridization studies. Our data show that fibroblasts are the only source of nidogen during early basal lamina formation. Although fibroblasts can synthesize nidogen and deposit it in the dermal matrix, no basal lamina will form unless they are recombined with keratinocytes. This suggests that the epidermis plays a major regulatory role in the production and assembly of nidogen into the basal lamina.

L9 ANSWER 8 OF 29 MEDLINE DUPLICATE 5
ACCESSION NUMBER: 95009530 MEDLINE
DOCUMENT NUMBER: 95009530 PubMed ID: 7925005
TITLE: Role of mesenchymal nidogen for epithelial morphogenesis in vitro.
AUTHOR: Ekblom P; Ekblom M; Fecker L; Klein G; Zhang H Y; Kadoya Y; Chu M L; Mayer U; Timpl R
CORPORATE SOURCE: Department of Animal Physiology, Uppsala University, Sweden.
CONTRACT NUMBER: AR 38923 (NIAMS)
SOURCE: DEVELOPMENT, (1994 Jul) 120 (7) 2003-14.
Journal code: 8701744. ISSN: 0950-1991.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199411
ENTRY DATE: Entered STN: 19941222
Last Updated on STN: 19941222
Entered Medline: 19941101

AB Recent biochemical studies suggested that the extracellular matrix protein nidogen is a binding molecule linking together basement membrane components. We studied its expression and role during development. By immunofluorescence and northern blotting, nidogen was found early during epithelial cell development of kidney and lung. Yet, in situ hybridization revealed that nidogen was not produced by epithelium but by the adjacent mesenchyme in both organs. Binding of mesenchymal nidogen to epithelial laminin may thus be a key event during epithelial development. This is supported by antibody perturbation experiments. **Antibodies** against the **nidogen** binding site on laminin B2 chain perturbed epithelial development in vitro in embryonic kidney and lung. Mesenchymal nidogen could be important for early stages of epithelial morphogenesis.

L9 ANSWER 9 OF 29 MEDLINE DUPLICATE 6
ACCESSION NUMBER: 95051016 MEDLINE
DOCUMENT NUMBER: 95051016 PubMed ID: 7962110
TITLE: Influence of nidogen complexed or not with laminin on attachment, spreading, and albumin and laminin B2 mRNA levels of rat hepatocytes.
AUTHOR: Levavasseur F; Mayer U; Guillouzo A; Clement B
CORPORATE SOURCE: Unite de Recherches Hepatologiques, INSERM U-49, Hopital Pontchaillou, Rennes, France.
SOURCE: JOURNAL OF CELLULAR PHYSIOLOGY, (1994 Nov) 161 (2) 257-66.
Journal code: 0050222. ISSN: 0021-9541.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199412
ENTRY DATE: Entered STN: 19950110
Last Updated on STN: 19950110

Entered Medline: 19941228

AB Nidogen/entactin is a Mr = 150,000 glycoprotein which is present within basement membranes in a noncovalent stable complex with laminin. We have studied the effects of nidogen/entactin complexed or not with laminin on attachment, spreading, and functions of adult rat hepatocytes in primary culture. Freshly isolated hepatocytes attached on either recombinant or EHS-derived nidogen, although to a lesser extent than on laminin/nidogen complex, laminin, and E8 and P1 fragments of laminin. Hepatocytes bound on a nidogen fragment bearing the N-terminal and rod-like domains but not on either the N-terminal globules or the rod-like domain which contains a RGD sequence. Attachment of hepatocytes on nidogen and laminin/**nidogen** complex was inhibited by anti-beta 1 integrin **antibodies**. Hepatocytes remained rounded on nidogen and laminin, whereas they rapidly spread on laminin/nidogen complex and collagen IV. Nidogen, laminin, and laminin/nidogen complex transiently maintained high steady-state albumin mRNA levels in cultured hepatocytes, but a decrease in albumin mRNA content was observed after 24 h, independently of the substrates. Actinomycin D and cycloheximide treatment indicated that the transient effect of these substrates on albumin expression was related to post-transcriptional mechanisms. Laminin B2 mRNAs were not detectable in freshly isolated hepatocytes but were expressed in 4 h hepatocyte cultures. After 24 h, a dramatic increase in the steady-state level of laminin B2 mRNA was found in hepatocytes cultured on nidogen and laminin/nidogen complex. This effect was slightly prevented in hepatocytes plated on laminin. These results show that interactions of hepatocytes with nidogen/entactin in vitro result only in a transient modulation of hepatocyte functions.

L9 ANSWER 10 OF 29 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.DUPLICATE 7

ACCESSION NUMBER: 1993:342697 BIOSIS
DOCUMENT NUMBER: PREV199396039697
TITLE: A single EGF-like motif of laminin is responsible for high affinity nidogen binding.
AUTHOR(S): Mayer, Ulrike; Nischt, Roswitha; Poeschl, Ernst; Mann, Karlheinz; Fukuda, Katsunori; Gerl, Martin; Yamada, Yoshihiko; Timpl, Rupert (1)
CORPORATE SOURCE: (1) Max-Planck-Inst. Biochem., D-8033 Martinsried Germany
SOURCE: EMBO (European Molecular Biology Organization) Journal, (1993) Vol. 12, No. 5, pp. 1879-1885. ISSN: 0261-4189.
DOCUMENT TYPE: Article
LANGUAGE: English

AB A major nidogen binding site of mouse laminin was previously localized to about three EGF-like repeats (Nos 3-5) of its B2 chain domain III (M.Gerl et al. (1991) Eur. J. Biochem., 202, 167). The corresponding cDNA was amplified by polymerase chain reaction and inserted into a eukaryotic expression vector tagged with a signal peptide. Stably transfected human kidney cell clones were shown to process and secrete the resulting fragment B2III3-5 in substantial quantities. It possessed high binding activity for recombinant nidogen in ligand assays, with an affinity comparable with that of authentic laminin fragments. In addition, complexes of B2III3-5 and nidogen could be effectively converted into a covalent complex by cross-linking reagents. Proteolytic degradation of the covalent complex demonstrated the association of BIII3-5 with a apprx 80 residue segment of nidogen domain G3 to which laminin binding has previously been attributed. The correct formation of most of the 12 disulfide bridges in B2III3-5 was indicated from its protease resistance and the complete loss of cross-reacting epitopes as well as of nidogen-binding activity after reduction and alkylation. Smaller fragments were prepared by the same recombinant procedure and showed that combinations of EGF-like repeats 3-4 and 4-5 and the single repeat 4 but not repeats 3 or 5 possess full nidogen-binding activity. This identifies repeat 4 as the only binding structure. The sequence of repeat 4 is well conserved in the human and in part in the Drosophila laminin B2 chain. It is further shown that **antibodies** against B2III3-5 inhibit laminin binding to **nidogen**, indicating that repeat 4 represents the only high affinity binding site of laminin.

L9 ANSWER 11 OF 29 MEDLINE DUPLICATE 8

ACCESSION NUMBER: 93146648 MEDLINE
DOCUMENT NUMBER: 93146648 PubMed ID: 8425764
TITLE: Myoepithelial and basement membrane antigens in benign and malignant human breast tumors.
AUTHOR: Guelstein V I; Tchypysheva T A; Ermilova V D; Ljubimov A V
CORPORATE SOURCE: Cancer Research Center, Russian Academy of Medical Sciences, Moscow.
SOURCE: INTERNATIONAL JOURNAL OF CANCER, (1993 Jan 21) 53 (2) 269-77. Journal code: 0042124. ISSN: 0020-7136.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199303
ENTRY DATE: Entered STN: 19930312
Last Updated on STN: 19930312
Entered Medline: 19930304

AB Serial cryostat sections of 160 human breast lesions and of 9 lymph-node metastases were studied by indirect immunofluorescence. We used monoclonal antibodies (MAbs) to lining-epithelium-specific keratin 8 and to myoepithelium-specific keratin 17 in combination with polyclonal and monoclonal **antibodies** to major basement membrane components, laminin, collagen type IV, entactin/**nidogen**, and large heparan sulfate proteoglycan (perlecan) core protein. Continuous basement membranes adjacent to a basal layer of keratin-17-positive myoepithelial cells were typical for normal, benign and in situ carcinomatous structures. In invasive and metastatic structures, always formed by keratin-8-positive tumor cells, basement membranes were found only rarely and with conspicuous fragmentations. This lack of basement membranes correlated with loss of myoepithelium identified by staining for keratin 17. In comedo structures of invasive ductal carcinomas and in papillary carcinomas, fibrovascular complexes with numerous blood vessels and deposition of basement membrane material were often seen in the stroma. Immunomorphological analysis of 41 cases of doubtful diagnosis at intra-operative biopsy was also performed. A combination of MAbs to keratins 8 and 17, and to basement membrane components, made it possible to distinguish between morphologically similar benign and malignant proliferations and to detect single-cell invasion of the stroma. This combination of antibodies may be recommended as an auxiliary immunomorphological tool for differential diagnosis of intra-operative breast biopsies in dubious cases.

L9 ANSWER 12 OF 29 MEDLINE DUPLICATE 9

ACCESSION NUMBER: 93238676 MEDLINE
DOCUMENT NUMBER: 93238676 PubMed ID: 8477687
TITLE: Ascidian entactin/nidogen. Implication of evolution by shuffling two kinds of cysteine-rich motifs.
AUTHOR: Nakae H; Sugano M; Ishimori Y; Endo T; Obinata T
CORPORATE SOURCE: Advanced Research Laboratory, Research and Development Center, Toshiba Corporation, Japan.
SOURCE: EUROPEAN JOURNAL OF BIOCHEMISTRY, (1993 Apr 1) 213 (1) 11-9. Journal code: 0107600. ISSN: 0014-2956.
PUB. COUNTRY: GERMANY: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-D14038; GENBANK-L09679; GENBANK-L09680; GENBANK-L09681; GENBANK-L09682; GENBANK-L09683;

GENBANK-X57950; GENBANK-X70793; GENBANK-X70999; GENBANK-X71000
ENTRY MONTH: 199305
ENTRY DATE: Entered STN: 19930611
Last Updated on STN: 20000303
Entered Medline: 19930521

AB Entactin/nidogen, a major component of the basement membrane, has a domain structure comprising three globular domains, and thread-like and rod-like domains connecting them. It contains six epidermal-growth-factor-(EGF)-like motifs and one thyroglobulin-like motif. In the present study, ascidian entactin/**nidogen** has been identified by a monoclonal **antibody** technique. We prepared anti-(ascidian entactin/nidogen)IgG, named anti-AsEnt1, then cloned the cDNA of ascidian entactin/nidogen using anti-AsEnt1 as a probe, and determined its entire sequence. Mainly because the deduced amino acid sequence exhibited high similarity to mouse entactin and human nidogen, and because the antigen localized in basement membrane of ascidian body-wall muscle, we have concluded that the antigen anti-AsEnt1 corresponds to the ascidian entactin/nidogen homologue. The deduced amino acid sequence of ascidian entactin/nidogen clearly showed that the ascidian homologue also has a domain structure. However, the ascidian homologue lacked the thread-like domain, and the rod-like domain differed from that of mouse entactin in composition, consisting of two kinds of cysteine-rich motifs, that is, the EGF-like motif and the thyroglobulin-like motif. These results suggest that entactin/nidogen have evolved by modifying the domains, especially by shuffling the two kinds of cysteine-rich motifs.

L9 ANSWER 13 OF 29 MEDLINE DUPLICATE 10
ACCESSION NUMBER: 92165419 MEDLINE
DOCUMENT NUMBER: 92165419 PubMed ID: 1371500
TITLE: Distribution of individual components of basement membrane in human colon polyps and adenocarcinomas as revealed by monoclonal antibodies.
AUTHOR: Ljubimov A V; Bartek J; Couchman J R; Kapuller L L; Veselov V V; Kovarik J; Perevoshchikov A G; Krutovskikh V A
CORPORATE SOURCE: All-Union Cancer Research Center, USSR AMS, Moscow.
CONTRACT NUMBER: AR36457 (NIAMS)
SOURCE: INTERNATIONAL JOURNAL OF CANCER, (1992 Feb 20) 50 (4) 562-6.
Journal code: 0042124. ISSN: 0020-7136.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199203
ENTRY DATE: Entered STN: 19920417
Last Updated on STN: 19980206
Entered Medline: 19920330

AB Double-label immunofluorescence was used to monitor basement-membrane composition and integrity in 22 human colon polyps, 36 adenocarcinomas and 2 metastases. Cryostat sections were stained with polyclonal anti-laminin anti-serum combined with monoclonal **antibodies** (MAbs) to all major basement-membrane components (laminin, entactin/**nidogen**, collagen type IV and large heparan sulfate proteoglycan), as well as to keratin 8. In all adenocarcinomas, including mucinous, basement membranes were altered more at the invasive front than in the parenchyma. The degree of this alteration was inversely correlated with the level of tumor differentiation. An uncoordinated loss of basement membrane components (dissociation of markers), previously described by us in rat colon adenocarcinomas, was also found in human tumors. In the great majority of adenocarcinomas a pronounced stromal reaction was seen. It was manifested by the presence of fibrillar deposits of basement-membrane components, mainly of collagen type IV and/or heparan sulfate proteoglycan. This reaction was never observed in polyps and may be derived from myofibroblasts reported to accumulate in colon cancer stroma. The combined use of antibodies to basement-membrane components and to a specific keratin may constitute an adequate immunohistochemical test for the presence of invasion, and may be useful in the histologic analysis of polyps, especially in dubious cases.

L9 ANSWER 14 OF 29 MEDLINE DUPLICATE 11
ACCESSION NUMBER: 94218359 MEDLINE
DOCUMENT NUMBER: 94218359 PubMed ID: 1344818
TITLE: Patterns of basement membrane laminin distribution in nonneoplastic and neoplastic thyroid tissue.
AUTHOR: Campo E; Perez M; Charonis A A; Axiotis C A; Merino M J
CORPORATE SOURCE: Laboratory of Pathology, National Institutes of Health, Bethesda, Maryland.
SOURCE: MODERN PATHOLOGY, (1992 Sep) 5 (5) 540-6.
Journal code: 8806605. ISSN: 0893-3952.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199405
ENTRY DATE: Entered STN: 19940606
Last Updated on STN: 19940606
Entered Medline: 19940524

AB Laminin, a major basement membrane component, is typically absent or partially lost around the epithelial elements of most invasive carcinomas. To evaluate the distribution of laminin in both primary and metastatic thyroid tumors, we studied 14 benign thyroid lesions (eight adenomas, two Graves' disease, two Hashimoto's thyroiditis, one adenomatous hyperplasia, one nodular goiter), 20 carcinomas (seven papillary, six tall cell variant, four follicular, three Hurthle), and eight metastases (five tall cell variant, three follicular) utilizing a polyclonal **antibody** against highly purified, **nidogen**-free laminin. All benign lesions showed positive, linear immunostaining along basement membranes. Partial loss or absence of laminin was seen in the solid areas of all types of thyroid carcinomas examined; well-differentiated papillary and follicular tumors, as well as papillary and follicular areas of more poorly differentiated neoplasms, maintained linear laminin immunostaining in the papillary cores beneath the epithelial cells and around follicles. A similar correlation between laminin deposition and architectural organization was seen in metastatic lesions. Hurthle cell carcinomas had a unique fragmented, pericellular immunostaining pattern around individual tumor cells, suggesting uncontrolled laminin synthesis. Our findings suggest that preservation of laminin production in thyroid tumors reflects their degree of differentiation and that absence of laminin correlates with lack of structural organization rather than reflecting invasive and metastatic potential.

L9 ANSWER 15 OF 29 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. DUPLICATE 12
ACCESSION NUMBER: 1993:95611 BIOSIS
DOCUMENT NUMBER: PREV199395050807
TITLE: Characterization of a natural human antibody with anti-galactosyl(alpha-1-2)galactose specificity that is present at high titers in chronic Trypanosoma cruzi infection.
AUTHOR(S): Avila, Jose Luis; Rojas, Miguel; Velaquez-Avila, Gladys
CORPORATE SOURCE: Inst. Biomed. Caracas, Venezuela, Hosp. de Ninos J. M. de los Rios, Caracas Venezuela
SOURCE: American Journal of Tropical Medicine and Hygiene, (1992) Vol. 47, No. 4, pp. 413-421.
ISSN: 0002-9637.
DOCUMENT TYPE: Article
LANGUAGE: English

AB An antibody reactive with the galactosyl(alpha-1-2)galactose (gal(alpha-1-2)gal) epitope was characterized in human sera by

enzyme-linked immunosorbent assay, red blood cell (RBC) and laminin absorption, and oligosaccharide inhibition. This antibody was found evenly distributed between the IgG and IgM classes and was present at high titers in the serum of all normal adults studied, but in 75% of children less than three years of age, it was observed at the lower limit of detection, and gradually increased to adult levels by the age of six. Although this antibody bound to gal(alpha-1-3)gal-linked synthetic antigens, it did not bind to the same residues present in rabbit, rat, and guinea pig RBC or in murine laminin or **nidogen**. These latter results, plus the fact that antigen-**antibody** binding was strongly blocked by gal(alpha-1-2)gal but not by methyl-alpha-galactopyranoside or melibiose, suggest that this antibody is indeed different from anti-gal(alpha-1-3)gal antibody. Anti-gal(alpha-1-2)gal antibody levels were significantly elevated in 66% of patients with chronic chagasic cardiomyopathy, but were not elevated in patients with different clinical forms of leishmaniasis, Trypanosoma rangeli-infected patients, or in patients with 15 other infectious and inflammatory diseases. Gal(alpha-1-2)gal antibodies did not absorb to intact T. cruzi parasites, but absorbed strongly to trypomastigote and epimastigote sonicates, suggesting some masking of reactive epitopes. Since antibody binding is blocked by gal(alpha-1-3)gal, previous results suggest that in chronic T. cruzi infection, at least three different antibody clones exist that react with gal(alpha-1-3)gal epitopes: anti-gal(alpha-1-3)gal IgG, anti-mannose (man)(alpha-1-3)gal or anti-man(beta-1-3)gal IgM, and anti-gal(alpha-1-2)gal IgM and IgG.

L9 ANSWER 16 OF 29 MEDLINE DUPLICATE 13

ACCESSION NUMBER: 92111677 MEDLINE
DOCUMENT NUMBER: 92111677 PubMed ID: 1370418
TITLE: American Leishmania spp. and Trypanosoma cruzi: galactosyl alpha(1-3) galactose epitope localization by colloidal gold immunocytochemistry and lectin cytochemistry.
AUTHOR: Bretana A; Avila J L; Contreras-Bretana M; Tapia F J
CORPORATE SOURCE: Secci+5Uon de Microscopia Electronica, Instituto de Biomedicina, Caracas, Venezuela.
SOURCE: EXPERIMENTAL PARASITOLOGY, (1992 Feb) 74 (1) 27-37. Journal code: 0370713. ISSN: 0014-4894.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199202
ENTRY DATE: Entered STN: 19920308
Last Updated on STN: 19960129
Entered Medline: 19920218

AB Patients with Chagas' disease or different clinical forms of leishmaniasis (cutaneous or visceral) have elevated galactosyl alpha (1-3)galactose antibodies. Using colloidal gold immunocytochemistry--monoclonal antibody gal-13 (specific for lipid-linked galactosyl alpha (1-3)galactose residues) and anti-**nidogen antibodies** and lectin cytochemistry (Bandeiraea simplicifolia IB4), both techniques specific for demonstrating galactosyl alpha (1-3)galactose residues--we have found terminal disaccharide residues on the Trypanosoma cruzi external surface of Vero cell-derived trypomastigotes but not in intact epimastigotes (although disrupted epimastigotes strongly stained), in the lips of the flagellar pocket, and on the parasitic side exactly opposite to the flagellar pocket in amastigote and promastigote forms of American Leishmania. These results resemble those obtained using anti-laminin antibodies in both trypanosomatids. In addition, results obtained with anti-**nidogen antibodies** seem to recognize in Trypanosoma cruzi and American Leishmania culture forms another different unknown terminal disaccharide. These results confirm the presence of terminal galactosyl alpha (1-3)galactose residues in both trypanosomatids, and that rabbit anti-laminin antibodies are indeed also recognizing galactosyl alpha (1-3)galactose residues as demonstrated for human circulating antibody. The presence of abundant galactosyl alpha (1-3)galactose residues on Trypanosomatid family members suggests a specific unknown role in parasite physiology for this terminal disaccharide.

L9 ANSWER 17 OF 29 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.DUPLICATE 14

ACCESSION NUMBER: 1990:518268 BIOSIS
DOCUMENT NUMBER: BA90:135544
TITLE: ULTRASTRUCTURAL LOCALIZATION OF THE CORE PROTEIN OF A BASEMENT MEMBRANE-SPECIFIC CHONDROITIN SULFATE PROTEOGLYCAN IN ADULT RAT SKIN.
AUTHOR(S): MCCARTHY K J; HORIGUCHI Y; COUCHMAN J R; FINE J-D
CORPORATE SOURCE: DEP. CELL BIOL. AND ANAT., VH 201 C BOX 803, UNIV. ALA. BIRMINGHAM, BIRMINGHAM, ALA. 35294, USA.
SOURCE: ARCH DERMATOL RES, (1990) 282 (6), 397-401. CODEN: ADREDL. ISSN: 0340-3696.
FILE SEGMENT: BA; OLD
LANGUAGE: English

AB Basement membranes are complex extracellular matrices present at epithelial/mesenchymal interfaces of tissues. The dermal-epidermal junction has been shown to contain numerous components, some of the most well known being laminin, types IV and VII collagens, heparin sulfate proteoglycan, fibronectin, and entactin/**nidogen**. In this paper we show, using core protein-specific **antibodies**, the presence of a newly described basement membrane-specific chondroitin sulfate proteoglycan at the epithelial/mesenchmal interval of adult rat skin. Ultrastructurally, this antigen was proven to reside primarily within the basal lamina, apparently concentrated in the lamina densa. In addition, some of the proteoglycan was also present beneath the lamina densa, associated with the reticular lamina collagen fibrils.

L9 ANSWER 18 OF 29 MEDLINE DUPLICATE 15

ACCESSION NUMBER: 90384093 MEDLINE
DOCUMENT NUMBER: 90384093 PubMed ID: 2119467
TITLE: Entactin: a possible auto-antigen in the pathogenesis of non-Goodpasture anti-GBM nephritis.
AUTHOR: Saxena R; Bygren P; Butkowski R; Wieslander J
CORPORATE SOURCE: Department of Nephrology, University Hospital of Lund, Sweden.
SOURCE: KIDNEY INTERNATIONAL, (1990 Aug) 38 (2) 263-72. Journal code: 0323470. ISSN: 0085-2538.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199010
ENTRY DATE: Entered STN: 19901122
Last Updated on STN: 19980206
Entered Medline: 19901026

AB It has recently been demonstrated that many patients with various types of glomerulonephritis have antibodies to the 6M guanidine-HCl extract of glomerular basement membrane (Bygren et al, Nephrol Dial Transplant 4:254-261, 1989). In the present study a 150 K protein was isolated from the guanidine extract of bovine glomerular basement membrane utilizing ion exchange and gel filtration chromatographic procedures. Amino acid analysis and size of the isolated protein revealed similarity to that of entactin/nidogen. The identity of this protein as entactin/**nidogen** was further suggested by its precipitation with two different **antibodies** in a radioimmunoassay and by its reaction with four different antibodies in a sandwich ELISA. Inhibition of the antibodies to 150 K by bovine entactin, which was isolated separately and sequenced for amino acids, confirmed the identity of the 150 K protein as entactin/nidogen. Furthermore, it was shown that about one third of those patients who show antibodies to the crude guanidine extract have circulating antibodies directed against entactin. This was further

confirmed by the competitive inhibition of antibodies to the crude guanidine extract in one of the positive serum by entactin in an ELISA inhibition and by immunoblotting experiments. These observations propose entactin as a possible non-Goodpasture glomerular basement membrane antigen that could be involved in the pathogenesis of certain forms of autoimmune glomerulonephritis (non-Goodpasture anti-GBM glomerulonephritis) in man. Most of these patients have a granular pattern of the immunoglobulin deposition along the glomerular basement membrane. This suggests the possibility that anti-GBM glomerulonephritis in human beings can have non-linear immunoglobulin deposits along the GBM.

L9 ANSWER 19 OF 29 MEDLINE DUPLICATE 16
ACCESSION NUMBER: 90118740 MEDLINE
DOCUMENT NUMBER: 90118740 PubMed ID: 2481931
TITLE: An improved immunofluorescence technique for the histological examination of blood vessel tissue.
AUTHOR: Kittelberger R; Davis P F; Stehbens W E
CORPORATE SOURCE: Malaghan Institute of Medical Research, Wellington School of Medicine, Wellington Hospital, New Zealand.
SOURCE: ACTA HISTOCHEMICA, (1989) 86 (2) 137-42.
Journal code: 0370320. ISSN: 0065-1281.
PUB. COUNTRY: GERMANY, EAST: German Democratic Republic
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199002
ENTRY DATE: Entered STN: 19900328
Last Updated on STN: 19960129
Entered Medline: 19900220

AB Autofluorescence of elastic fibres in blood vessel samples is a common interference with the specific fluorescence of FITC-conjugated antibodies. Counterstaining with eriochrome black T changed the yellow-green colour of elastic fibres to dark red, thus turning a disturbing feature into a useful reference background. A second counterstain, p-phenylenediamine, visualized cell nuclei as an amber colour. To demonstrate the improvement of this staining technique, cryosections from blood vessel samples, derived from control veins, arteries and experimental aneurysms of different ages (15 to 99 month old) in 5 sheep, were stained with **antibodies** against procollagen III, collagen type IV, laminin, and **nidogen**. The specific distribution of these connective tissue components could now be related to the location of the elastic fibres and the cells (cell nuclei).

L9 ANSWER 20 OF 29 MEDLINE
ACCESSION NUMBER: 90143096 MEDLINE
DOCUMENT NUMBER: 90143096 PubMed ID: 2482635
TITLE: [Structure and antigenicity of the glomerular basement membrane].
Aufbau und Antigenitat der glomerularen Basalmembran.
AUTHOR: Weber M
SOURCE: VERHANDLUNGEN DER DEUTSCHEN GESELLSCHAFT FUR PATHOLOGIE, (1989) 73 6-12. Ref: 38
Journal code: 7503704. ISSN: 0070-4113.
PUB. COUNTRY: GERMANY, WEST: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: German
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199003
ENTRY DATE: Entered STN: 19900328
Last Updated on STN: 19960129
Entered Medline: 19900312

AB The glomerular basement membrane is a complex extracellular matrix formed of various molecules which build a supramolecular network. The major structural components are collagen IV, laminin, heparan sulfate proteoglycan, and nidogen/entactin. Cross-reacting **antibodies** against laminin, **nidogen**, and collagen IV may occur after several infectious diseases. They are however of doubtful pathogenetic significance. The pathogenetic relevant autoantibodies in Goodpasture's syndrome and rapidly progressive glomerulonephritis with linear immunofluorescence pattern are directed against epitopes which are located on the collagenase resistant C-terminal globule NC1 of collagen IV. The human NC1 globule appears as a hexamer which dissociates into monomers and dimers under various experimental conditions. Dissociation is paralleled by a significant increase in available epitopes. Immunisation with the dissociated NC1 globule initiates a pulmo-renal syndrome in rabbits similar to the human Goodpasture's syndrome. In hereditary nephritis one of the alpha-chains which form the triple-helix of collagen IV seems to be altered within the NC1 region. This may possibly explain the typical morphologic findings in this disease as well as the reduced binding of antiglomerular basement membrane antibodies to basement membranes of kidneys in Alport's syndrome.

L9 ANSWER 21 OF 29 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1988:567788 CAPLUS
DOCUMENT NUMBER: 109:167788
TITLE: High resolution immunoelectron microscopic localization of functional domains of laminin, nidogen, and heparan sulfate proteoglycan in epithelial basement membrane of mouse cornea reveals different topological orientations
AUTHOR(S): Schittny, Johannes C.; Timpl, Rupert; Engel, Juergen
CORPORATE SOURCE: Biocent., Univ. Basel, Basel, CH-4056, Switz.
SOURCE: Journal of Cell Biology (1988), 107(4), 1599-610
CODEN: JCLBA3; ISSN: 0021-9525
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Thin and ultrathin cryosections of mouse cornea were labeled with affinity-purified antibodies directed against either laminin, its central segments (domain 1), the end of its long arm (domain 3), the end of one of its short arms (domain 4), nidogen, or low-d. heparan sulfate proteoglycan. All basement membrane proteins were detected by indirect immunofluorescence exclusively in the epithelial basement membrane, in Descemet's membrane, and in small amorphous plaques located in the stroma. Immunoelectron microscopy with the protein A-Au technique demonstrated laminin domain 1 and nidogen in a narrow segment of the lamina densa at the junction to the lamina lucida within the epithelial basement membrane. Domain 3 showed 3 preferred locations at both the cellular and stromal boundaries of the epithelial basement membrane and in its center. Domain 4 was located predominantly in the lamina lucida and the adjacent half of the lamina densa. The low-d. heparan sulfate proteoglycan was found all across the basement membrane, showing a similar uniform distribution as with antibodies against the whole laminin mol. In Descemet's membrane an even distribution was found with all these antibodies. Hence, within the epithelial basement membrane the center of the laminin mol. is located near the lamina densa/lamina lucida junction and its long arm favors 3 major orientations. One is close to the cell surface indicating binding to a cell receptor, whereas the other 2 are directed to internal matrix structures. The apparent codistribution of laminin domain 1 and nidogen agrees with biochem. evidence that nidogen binds to this domain.

L9 ANSWER 22 OF 29 MEDLINE DUPLICATE 17
ACCESSION NUMBER: 88151991 MEDLINE
DOCUMENT NUMBER: 88151991 PubMed ID: 3126070
TITLE: Analysis of degradation of the basement membrane protein **nidogen**, using a specific monoclonal **antibody**.
AUTHOR: Dziadek M; Clements R; Mitrangas K; Reiter H; Fowler K
CORPORATE SOURCE: Murdoch Institute for Research into Birth Defects, Royal

Children's Hospital, Parkville, Victoria, Australia.
SOURCE: EUROPEAN JOURNAL OF BIOCHEMISTRY, (1988 Feb 15) 172 (1) 219-25.
Journal code: 0107600. ISSN: 0014-2956.
PUB. COUNTRY: GERMANY, WEST: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198804
ENTRY DATE: Entered STN: 19900308
Last Updated on STN: 19900308
Entered Medline: 19880419

AB A monoclonal **antibody** was produced against purified **nidogen** extracted from a mouse basement-membrane-producing tumor. This antibody reacted with a determinant on Nd-40, a rod which separates the globular domains of nidogen. Antigenicity depends on intrachain disulfide bonds within this rod. The monoclonal **antibody** was used to detect **nidogen** fragments after proteolytic cleavage of isolated nidogen, and nidogen complexed to laminin. The data indicate that thrombin and thermolysin generated very different patterns of degradation, but in both cases no differences were found between isolated and complexed nidogen. In contrast, nidogen in the laminin-nidogen complex was much less degraded by trypsin than isolated nidogen, indicating that an interaction between these basement membrane components reduces the susceptibility of nidogen to trypsin digestion. Immunofluorescent studies, using the monoclonal antibody on sections of the EHS tumor after proteolytic digestion, showed that the retention or disappearance of the Nd-40 determinant correlated with the in vitro digestion pattern of the laminin-nidogen complex.

L9 ANSWER 23 OF 29 SCISEARCH COPYRIGHT 2003 ISI (R)
ACCESSION NUMBER: 88:109325 SCISEARCH
THE GENUINE ARTICLE: M2364
TITLE: ANALYSIS OF DEGRADATION OF THE BASEMENT-MEMBRANE PROTEIN **NIDOGEN**, USING A SPECIFIC MONOCLONAL-**ANTIBODY**
AUTHOR: DZIADEK M (Reprint); CLEMENTS R; MITRANGAS K; REITER H; FOWLER K
CORPORATE SOURCE: ROYAL CHILDRENS HOSP, MURDOCH INST RES BIRTH DEFECTS, PARKVILLE, VIC 3052, AUSTRALIA
COUNTRY OF AUTHOR: AUSTRALIA
SOURCE: EUROPEAN JOURNAL OF BIOCHEMISTRY, (1988) Vol. 172, No. 1, pp. 219-225.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: ENGLISH
REFERENCE COUNT: 42

L9 ANSWER 24 OF 29 MEDLINE DUPLICATE 18
ACCESSION NUMBER: 88139674 MEDLINE
DOCUMENT NUMBER: 88139674 PubMed ID: 2449451
TITLE: Serological activity against galactosyl-alpha(1-3)galactose in sera from patients with several kinetoplastida infections.
AUTHOR: Avila J L; Rojas M; Towbin H
CORPORATE SOURCE: Instituto de Biomedicina, Caracas, Venezuela.
SOURCE: JOURNAL OF CLINICAL MICROBIOLOGY, (1988 Jan) 26 (1) 126-32.
Journal code: 7505564. ISSN: 0095-1137.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198804
ENTRY DATE: Entered STN: 19900308
Last Updated on STN: 19900308
Entered Medline: 19880407

AB Using rabbit erythrocyte-derived neutral glycosphingolipids enriched for a defined ceramide pentasaccharide as antigens, we have detected elevated anti-galactosyl-alpha(1-3)galactose (anti-G alpha G) antibody values in patients with American cutaneous leishmaniasis (ACL), chronic Chagas' disease, and Trypanosoma rangeli infections compared with normal subjects or with patients suffering from any of 15 other infectious diseases. The specificity of the G alpha G antibodies was determined by inhibition enzyme-linked immunosorbent assays, which revealed that several alpha-galactosyl- but not beta-galactosyl-bearing sugars blocked absorption of G alpha G antibodies to the specific antigen used. G alpha G antibodies were mainly distributed between immunoglobulin classes G and M in three Kinetoplastida infections studied, with a lower increase in reactivity detected in immunoglobulin A. Absorption of highly reactive G alpha G **antibodies** with purified murine laminin and **nidogen**, two basement membrane proteins, almost abolished G alpha G reactivity, suggesting the identity of anti-G alpha G with laminin and **nidogen antibodies** previously reported as elevated in Kinetoplastida infections. In ACL, G alpha G antibodies were detected in 71% of patients having skin lesions with a clinical evolution time of 0.5 month. This percentage increased with the time of evolution of skin lesions, reaching 93% in lesions older than 3 months, and tended to decrease inversely to the induration diameter in the skin leishmanin test. It is proposed that similar epitopes may exist on kinetoplast protozoa and that the determination of G alpha G antibodies may be a highly sensitive assay for the detection of humoral responses to Kinetoplastida infections.

L9 ANSWER 25 OF 29 MEDLINE DUPLICATE 19
ACCESSION NUMBER: 87308118 MEDLINE
DOCUMENT NUMBER: 87308118 PubMed ID: 3114248
TITLE: The cellular interactions of laminin fragments. Cell adhesion correlates with two fragment-specific high affinity binding sites.
AUTHOR: Aumailley M; Nurcombe V; Edgar D; Paulsson M; Timpl R
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (1987 Aug 25) 262 (24) 11532-8.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198709
ENTRY DATE: Entered STN: 19900305
Last Updated on STN: 19970203
Entered Medline: 19870930

AB The molecular interactions of laminin with several tumor cell lines and skin fibroblasts were investigated by radioligand binding studies and cell attachment assays using laminin, the laminin-**nidogen** complex, and laminin fragments as substrates and also domain-specific **antibodies** as inhibitors of cell attachment. The majority of cells showed a dual binding pattern for fragments 1 and 8 which originate from short-arm or long-arm structures of laminin, respectively. Both of these fragments in solution bind to suspended cells with high affinity (KD = 1-10 nM), with the receptor numbers for each fragment depending on the cell type. Competition studies and independent variation of receptor numbers demonstrated that the cell-binding structures on each fragment are different, implicating the existence of two distinct cellular receptors for laminin. The ability of these fragments to act as substrates for cell adhesion correlated with the presence of high affinity binding sites on the cells. However, only antibodies to fragment 8 were able to block cell adhesion to laminin, despite the presence of binding sites for fragment 1. A few cells had very low numbers of high affinity receptors for either fragment 1 or 8. The latter cell type was used to demonstrate that complex formation between laminin and nidogen, which binds to fragment 1 structures, reduces the potential of laminin for cell binding.

L9 ANSWER 26 OF 29 MEDLINE DUPLICATE 20
ACCESSION NUMBER: 88136304 MEDLINE
DOCUMENT NUMBER: 88136304 PubMed ID: 2449305
TITLE: **Antibodies** to basement membrane proteins **nidogen** and laminin in sera from streptococcal-related diseases and juvenile rheumatoid arthritis patients.
AUTHOR: Avila J L; Rojas M; Velazquez-Avila G; Rieber M
CORPORATE SOURCE: Instituto de Biomedicina, Caracas, Venezuela.
SOURCE: CLINICAL AND EXPERIMENTAL IMMUNOLOGY, (1987 Dec) 70 (3) 555-61.
Journal code: 0057202. ISSN: 0009-9104.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198803
ENTRY DATE: Entered STN: 19900308
Last Updated on STN: 19900308
Entered Medline: 19880328

AB Using the ELISA technique, **antibodies** against two different basement proteins, laminin and **nidogen** (ALNA), were determined in 226 children suffering from one of 37 different inflammatory or infectious diseases. These included 80 patients with streptococcal infection and 40 with juvenile rheumatoid arthritis. Forty-eight percent of the streptococcus-infected patients (or 75% of those in the acute phase) and 60% of juvenile rheumatoid arthritis patients had significantly elevated ALNA levels compared with healthy controls. Interestingly 10 adult rheumatoid arthritis patients displayed normal ALNA levels, suggesting a particular immune process occurring in children affected by juvenile rheumatoid arthritis. By means of periodate oxidation and glycosidase treatments we have shown that ALNA positive sera recognized terminal alpha-galactose as the reactive epitope.

L9 ANSWER 27 OF 29 MEDLINE DUPLICATE 21
ACCESSION NUMBER: 87034242 MEDLINE
DOCUMENT NUMBER: 87034242 PubMed ID: 2429987
TITLE: **Antibodies** to basement membrane protein **nidogen** in Chagas' disease and American cutaneous leishmaniasis.
AUTHOR: Avila J L; Rojas M; Velazquez-Avila G; von der Mark H; Timpl R
SOURCE: JOURNAL OF CLINICAL MICROBIOLOGY, (1986 Nov) 24 (5) 775-8.
Journal code: 7505564. ISSN: 0095-1137.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198612
ENTRY DATE: Entered STN: 19900302
Last Updated on STN: 19900302
Entered Medline: 19861216

AB About 50 to 70% of sera from patients with American cutaneous leishmaniasis and chronic Chagas' disease possessed **antibodies** which reacted in enzyme and radioimmunoassays with **nidogen** obtained from a tumor basement membrane. The antibodies were of the immunoglobulin M and G classes in acute American cutaneous leishmaniasis but mainly of the immunoglobulin G class in chronic Chagas' disease. Similar antibodies could not be detected in patients suffering from a variety of other infectious or inflammatory diseases when compared with healthy control groups. Inhibition and immunoadsorption studies indicated a close relationship of epitopes recognized by patients' **antibodies** on **nidogen** and on another basement membrane protein, laminin. Since rabbit antisera to both proteins do not cross-react, a special nature of the epitopes involved in the reaction with patient sera is suggested. Similar epitopes may exist on various forms of Leishmania or Trypanosoma protozoa.

L9 ANSWER 28 OF 29 MEDLINE DUPLICATE 22
ACCESSION NUMBER: 86005830 MEDLINE
DOCUMENT NUMBER: 86005830 PubMed ID: 2995165
TITLE: Expression of nidogen and laminin in basement membranes during mouse embryogenesis and in teratocarcinoma cells.
AUTHOR: Dziadek M; Timpl R
SOURCE: DEVELOPMENTAL BIOLOGY, (1985 Oct) 111 (2) 372-82.
Journal code: 0372762. ISSN: 0012-1606.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198510
ENTRY DATE: Entered STN: 19900321
Last Updated on STN: 19900321
Entered Medline: 19851029

AB Nidogen and laminin were localized at preimplantation stages of mouse development by immunofluorescence. Laminin was already present on the cell surface at the 2-cell stage, while nidogen was first detectable on compacted 8- to 16-cell stage morulae. Nidogen and laminin colocalized at the blastocyst stage and in postimplantation basement membranes. Immunoblot analyses of tissue extracts and cell culture media indicated the 150-kDa form of nidogen as the largest and predominant form in all tissues examined. Radiolabeled **nidogen** and laminin synthesized by Reichert's membrane were coprecipitated by **antibodies** against each antigen, indicating complex formation in situ. Equimolar amounts of laminin and nidogen were determined in 6 M guanidine X HCl extracts of tissues by radioimmunoassays, further indicating stoichiometric complexes. However, lower levels of nidogen than laminin were found in tissue and cell culture media. A less than 2-fold increase in nidogen was found when F9 cells were stimulated to differentiate with retinoic acid and dibutyryl cAMP, compared to a 30-fold increase in laminin secretion.

L9 ANSWER 29 OF 29 MEDLINE DUPLICATE 23
ACCESSION NUMBER: 84108344 MEDLINE
DOCUMENT NUMBER: 84108344 PubMed ID: 6420150
TITLE: Nidogen: a new, self-aggregating basement membrane protein.
AUTHOR: Timpl R; Dziadek M; Fujiwara S; Nowack H; Wick G
SOURCE: EUROPEAN JOURNAL OF BIOCHEMISTRY, (1983 Dec 15) 137 (3) 455-65.
Journal code: 0107600. ISSN: 0014-2956.
PUB. COUNTRY: GERMANY, WEST: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198403
ENTRY DATE: Entered STN: 19900319
Last Updated on STN: 19900319
Entered Medline: 19840301

AB Nidogen was purified from a mouse tumor basement membrane where it accounted for 2-3% of the total proteins. It was isolated as two forms (A and B) of a monomer (Mr = 80000) each consisting of a single polypeptide chain folded into a globular head connected to a small tail. The B form of the monomer was shown to be capable of aggregating into a nest-like structure (Mr greater than 250000). A smaller form (Mr = 45000) was observed in some of the extracts. The amino acid composition of nidogen was different to that of other basement membrane proteins. It contained about 10% carbohydrate, with N-linked and O-linked oligosaccharide chains in similar proportions. Isoelectrofocussing demonstrated a limited heterogeneity of nidogen with pI in the range 6.5 - 7. Monomeric nidogen failed to interact with other basement membrane components and heparin.

Aggregation could be induced by limited proteolysis and was reversed by detergents or high salt concentrations. Together with the observation that most of the nidogen could be solubilized only after destroying the collagenous matrix, the data indicate that aggregation of nidogen reflects an activity involved in matrix assembly. Specific **antibodies** raised against **nidogen** did not distinguish between the monomeric and aggregated form of the protein but showed that the fragment was antigenically deficient. These antibodies did not cross-react with collagen type IV, laminin, entactin and heparansulfate proteoglycan. Immunofluorescence staining and absorption studies demonstrated that nidogen is a common component of authentic basement membranes. Larger forms of nidogen (Mr about 100000 and 150000) were found in organ cultures of Reichert's membrane suggesting that it is synthesized in precursor forms.

```
=> s psi (10a) antibod?
L10         274 PSI (10A) ANTIBOD?

=> s l10 and plexin
L11           0 L10 AND PLEXIN

=> s l10 and semaphorin?
L12           1 L10 AND SEMAPHORIN?

=> d 1

L12    ANSWER 1 OF 1      PCTFULL   COPYRIGHT 2003 Univentio
AN     2000037483 PCTFULL  ED 20020515
TIEN   PROTEIN-PROTEIN INTERACTIONS IN NEURODEGENERATIVE DISORDERS
TIFR   INTERACTIONS PROTEINE-PROTEINE DANS LES TROUBLES NEURODEGENERATIFS
IN     ROCH, Jean-Marc;
       BARTEL, Paul, L.
PA     MYRIAD GENETICS, INC.
LA     English
DT     Patent
PI     WO 2000037483         A1 20000629
DS     W:           AE AL AM AT AU AZ BA BB BG BR BY CA CH CN CR CU CZ DE DK DM
                    EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ
                    LC LK LR LS LT LU LV MA MD MG MK MN MW MX NO NZ PL PT RO RU
                    SD SE SG SI SK SL TJ TM TR TT TZ UA UG UZ VN YU ZA ZW GH GM
                    KE LS MW SD SL SZ TZ UG ZW AM AZ BY KG KZ MD RU TJ TM AT BE
                    CH CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE BF BJ CF CG
                    CI CM GA GN GW ML MR NE SN TD TG
AI     WO 1999-US30396       A  19991221
PRAI   US 1998-60/113,534       19981222
       US 1999-60/124,120       19990312
       US 1999-60/141,243       19990630
ICM    C07K001-00
ICS    C07K014-00; C07K017-00; A01K067-00; A01K067-033; A01K067-027;
       C12Q001-68; G01N033-53; G01N033-567; C12N005-00; C12N005-02


=> d kwic

L12    ANSWER 1 OF 1      PCTFULL   COPYRIGHT 2003 Univentio

DETD . . . that links the cytoskeleton to the
       extracellular matrix. In another entry, mouse GIPC is called SemcapI
       (accession number
       AF061263), which stands for semaphorin F cytoplasmic domain
       associated protein I . Thus, GIPC
       is also thought to interact with sernaphorin F, and therefore, it is. .
       .
```

,NO
EXAMPLE 39
Generation of Polyclonal **Antibody** against **PSI**
-FKBP25 Complex
As shown above, APP interacts with FKBP25 to form a complex. A complex of the two
proteins is prepared, e.g.,. . .

EXAMPLE 40
Generation of Monoclonal **Antibodies** Specific for **PSI**
-FKBP25 Complex
Monoclonal **antibodies** are generated according to the following protocol. Mice are
immunized with immunogen comprising PSI-FKBP25 complexes conjugated to keyhole limpet
hemocyanin using glutaraldehyde. . .
. . .
culture plates. Individual wells are
examined for growth, and the supernatants of wells with growth are
tested for the presence of **PSI** -
FKBP25 complex-specific **antibodies** by ELISA or RIA using
PSI-FKBP25 corn lex as target
p
protein. Cells in positive wells are expanded and subeloned to establish
and confirm monoclonality.
. . .
grown as ascites in mice or in a
hollow fiber system to produce sufficient quantities of antibodies for
characterization and assay
development. **Antibodies** are tested for binding to **PSI**
alone or to FKBP25 alone, to determine
which are specific for the PSI -FKBP2 5 complex as opposed to those
that. . .

```
=> d his

     (FILE 'HOME' ENTERED AT 13:34:50 ON 24 APR 2003)

     FILE 'MEDLINE, CANCERLIT, BIOSIS, CONFSCI, CAPLUS, EMBASE, USPATFULL,
     PCTFULL, SCISEARCH' ENTERED AT 13:35:24 ON 24 APR 2003
L1          713 S NIDOGEN AND ANTIBOD?
L2          407 DUP REM L1 (306 DUPLICATES REMOVED)
L3          244 S L2 NOT PY=>2000
L4           45 S L3 AND FUSION
L5           10 S L4 AND HUMANIZE?
L6            9 S L5 AND SINGLE (A) CHAIN
L7          142 S NIDOGEN (10A) ANTIBOD?
L8          101 S L7 NOT PY=>1999
L9           29 DUP REM L8 (72 DUPLICATES REMOVED)
L10         274 S PSI (10A) ANTIBOD?
L11           0 S L10 AND PLEXIN
L12           1 S L10 AND SEMAPHORIN?

=> s l10 and integrin
L13           5 L10 AND INTEGRIN

=> d 1-5

L13  ANSWER 1 OF 5  USPATFULL
AN     2002:48017  USPATFULL
TI     NUCLEIC ACID ENCODING FELINE CD86
IN     COLLISSON, ELLEN W., COLLEGE STATION, TX, UNITED STATES
       HASH, STEPHEN M., AUSTIN, TX, UNITED STATES
       CHOI, INSOO, COLLEGE STATION, TX, UNITED STATES
```

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PI     US 2002028208      A1   20020307
AI     US 1999-303510     A1   19990430 (9)
PRAI   US 1998-83869P      19980501 (60)
DT     Utility
FS     APPLICATION
LN.CNT 4890
INCL   INCLM: 424/188.100
       INCLS: 536/023.500
NCL    NCLM:  424/188.100
       NCLS:  536/023.500
IC     [7]
       ICM: A61K039-21
       ICS: C07H021-04
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L13    ANSWER 2 OF 5     PCTFULL   COPYRIGHT 2003 Univentio
AN     2002022862 PCTFULL  ED 20020705  EW 200212
TIEN   METHODS FOR IDENTIFYING MODULATORS OF PROTEIN INTERACTIONS
TIFR   METHODES D'IDENTIFICATION DE MODULATEURS D'INTERACTIONS DE PROTEINES
IN     HILES, Ian, Donald, c/o Glaxo SmithKline plc, Gunnels Wood Road,
       Stevenage, Hertfordshire SG1 2NY, GB [GB, GB];
       ELLIS, Christine, c/o Glaxo SmithKline plc, Gunnels Wood Road,
       Stevenage, Hertfordshire SG1 2NY, GB [GB, GB]
PA     GLAXO GROUP LIMITED, Glaxo Wellcome House, Berkeley Avenue, Greenford,
       Middlesex UB6 0NN, GB [GB, GB], for all designates States except US;
       HILES, Ian, Donald, c/o Glaxo SmithKline plc, Gunnels Wood Road,
       Stevenage, Hertfordshire SG1 2NY, GB [GB, GB], for US only;
       ELLIS, Christine, c/o Glaxo SmithKline plc, Gunnels Wood Road,
       Stevenage, Hertfordshire SG1 2NY, GB [GB, GB], for US only
AG     WOODS, Geoffrey, Corlett, J.A. Kemp & Co., 14 South Square, Gray's Inn,
       London WC1R 5JJ, GB
LAF    English
LA     English
DT     Patent
PI     WO 2002022862        A2 20020321
DS     W:          AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ
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                   KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ
                   NO NZ PH PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG
                   US UZ VN YU ZA ZW
       RW (ARIPO): GH GM KE LS MW MZ SD SL SZ TZ UG ZW
       RW (EAPO):  AM AZ BY KG KZ MD RU TJ TM
       RW (EPO):   AT BE CH CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE TR
       RW (OAPI):  BF BJ CF CG CI CM GA GN GQ GW ML MR NE SN TD TG
AI     WO 2001-GB4094       A  20010912
PRAI   GB 2000-0022333.9      20000912
ICM    C12Q001-48
ICS    C12Q001-37; A61K035-00

L13    ANSWER 3 OF 5     PCTFULL   COPYRIGHT 2003 Univentio
AN     2001091792 PCTFULL  ED 20020826
TIEN   THERAPEUTIC IMMUNE CONDITION COMPOSITIONS AND METHODS
TIFR   COMPOSITIONS THERAPEUTIQUES DESTINEES A DES ETATS IMMUNITAIRES ET
       PROCEDE ASSOCIES
IN     MADIYALAKAN, Ragupathy;
       NOUJAIM, Antoine, A.;
       LEVEUGLE, Beatrice
PA     ALTAREX CORP.
DT     Patent
PI     WO 2001091792        A2 20011206
DS     W:          AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CU CZ DE
                   DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG
                   KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ
                   PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG UZ VN YU
                   ZA ZW GH GM KE LS MW MZ SD SL SZ TZ UG ZW AM AZ BY KG KZ MD
                   RU TJ TM AT BE CH CY DE DK ES FI FR GB GR IE IT LU MC NL PT
                   SE BF BJ CF CG CI CM GA GN GW ML MR NE SN TD TG
AI     WO 2000-IB2077       A  20000825
PRAI   US 1999-60/150,652     19990825
ICM    A61K039-395

L13    ANSWER 4 OF 5     PCTFULL   COPYRIGHT 2003 Univentio
AN     2000037483 PCTFULL  ED 20020515
TIEN   PROTEIN-PROTEIN INTERACTIONS IN NEURODEGENERATIVE DISORDERS
TIFR   INTERACTIONS PROTEINE-PROTEINE DANS LES TROUBLES NEURODEGENERATIFS
IN     ROCH, Jean-Marc;
       BARTEL, Paul, L.
PA     MYRIAD GENETICS, INC.
LA     English
DT     Patent
PI     WO 2000037483        A1 20000629
DS     W:          AE AL AM AT AU AZ BA BB BG BR BY CA CH CN CR CU CZ DE DK DM
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                   LC LK LR LS LT LU LV MA MD MG MK MN MW MX NO NZ PL PT RO RU
                   SD SE SG SI SK SL TJ TM TR TT TZ UA UG UZ VN YU ZA ZW GH GM
                   KE LS MW SD SL SZ TZ UG ZW AM AZ BY KG KZ MD RU TJ TM AT BE
                   CH CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE BF BJ CF CG
                   CI CM GA GN GW ML MR NE SN TD TG
AI     WO 1999-US30396      A  19991221
PRAI   US 1998-60/113,534     19981222
       US 1999-60/124,120     19990312
       US 1999-60/141,243     19990630
ICM    C07K001-00
ICS    C07K014-00; C07K017-00; A01K067-00; A01K067-033; A01K067-027;
       C12Q001-68; G01N033-53; G01N033-567; C12N005-00; C12N005-02

L13    ANSWER 5 OF 5     PCTFULL   COPYRIGHT 2003 Univentio
AN     1999057271 PCTFULL  ED 20020515
TIEN   FELINE CD80, FELINE CD86, FELINE CD28, AND FELINE CTLA-4 NUCLEIC ACID
       AND POLYPEPTIDES
TIFR   ACIDE NUCLEIQUE ET POLYPEPTIDES DES CD80, CD86, CD28 ET CTLA-4 FELINS
IN     COLLISON, Ellen, W.;
       HASH, Stephen, M.;
       CHOI, InSou
PA     THE TEXAS A & M SYSTEM
LA     English
DT     Patent
PI     WO 9957271           A2 19991111
DS     W:          AE AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES
                   FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK
                   LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG
                   SI SK SL TJ TM TR TT UA UG UZ VN YU ZA ZW GH GM KE LS MW SD
                   SL SZ UG ZW AM AZ BY KG KZ MD RU TJ TM AT BE CH CY DE DK ES
                   FI FR GB GR IE IT LU MC NL PT SE BF BJ CF CG CI CM GA GN GW
                   ML MR NE SN TD TG
AI     WO 1999-US9502       A  19990430
PRAI   US 1998-09/071,699     19980501
ICM    C12N015-12
ICS    C12N001-21; C12N005-10; C07K014-705; A61K039-21; A61K039-12;
       A61K038-17; A61K039-39


=> s antibod? and integrin
L14       45797 ANTIBOD? AND INTEGRIN

=> s l14 and single (a) chain
L15        3999 L14 AND SINGLE (A) CHAIN

=> s l15 and humanize?
L16        2934 L15 AND HUMANIZE?
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=> s l16 and fusion
L17         2832 L16 AND FUSION

=> dup rem l17
PROCESSING IS APPROXIMATELY  24% COMPLETE FOR L17
PROCESSING IS APPROXIMATELY  45% COMPLETE FOR L17
PROCESSING IS APPROXIMATELY  69% COMPLETE FOR L17
PROCESSING IS APPROXIMATELY  84% COMPLETE FOR L17
PROCESSING COMPLETED FOR L17
L18         2831 DUP REM L17 (1 DUPLICATE REMOVED)

=> s l17 not py=>1999
'1999' NOT A VALID FIELD CODE
L19         171 L17 NOT PY=>1999

=> d 1-30

L19  ANSWER 1 OF 171  USPATFULL
AN      1998:162339  USPATFULL
TI      Murine and humanizer 23F2G antibodies and cell lines
        expressing said antibodies
IN      Rose, Lynn M., Seattle, WA, United States
PA      ICOS Corporation, Bothell, WA, United States (U.S. corporation)
        Board of Regents of the University of Washington, Seattle, WA, United
        States (U.S. corporation)
PI      US 5854070                19981229
AI      US 1997-785571            19970121 (8)
RLI     Continuation of Ser. No. US 1995-396089, filed on 28 Feb 1995, now
        abandoned which is a continuation of Ser. No. US 1993-94535, filed on 16
        Jul 1993, now abandoned which is a continuation-in-part of Ser. No. US
        1993-60699, filed on 10 May 1993, now abandoned which is a continuation
        of Ser. No. US 1992-915068, filed on 16 Jul 1992, now abandoned
DT      Utility
FS      Granted
LN.CNT 1524
INCL    INCLM: 435/343.200
        INCLS: 435/343.000; 435/343.100; 435/326.000; 435/328.000; 435/346.000;
               435/358.000; 435/334.000; 530/387.100; 530/387.300; 530/388.100;
               530/388.200; 530/388.220; 530/388.700; 530/388.730; 530/388.750
NCL     NCLM:  435/343.200
        NCLS:  435/326.000; 435/328.000; 435/334.000; 435/343.000; 435/343.100;
               435/346.000; 435/358.000; 530/387.100; 530/387.300; 530/388.100;
               530/388.200; 530/388.220; 530/388.700; 530/388.730; 530/388.750
IC      [6]
        ICM: C07K016-18
        ICS: C07K016-28; C12N005-12
EXF     435/70.21; 435/172.2; 435/334; 435/343.2; 530/387.1; 530/388.2;
        530/388.73
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L19  ANSWER 2 OF 171  USPATFULL
AN      1998:162259  USPATFULL
TI      Decorin binding protein compositions and methods of use
IN      Guo, Betty, Houston, TX, United States
        Hook, Magnus, Houston, TX, United States
PA      The Texas A & M University System, College Station, TX, United States
        (U.S. corporation)
PI      US 5853987                19981229
AI      US 1996-589711            19960122 (8)
RLI     Continuation-in-part of Ser. No. US 1995-427023, filed on 24 Apr 1995,
        now abandoned
DT      Utility
FS      Granted
LN.CNT 4684
INCL    INCLM: 435/006.000
        INCLS: 536/022.100; 536/023.100; 536/023.700; 536/024.330; 536/025.320;
               435/320.100; 435/091.200; 435/006.000; 530/350.000
NCL     NCLM:  435/006.000
        NCLS:  435/091.200; 435/320.100; 530/350.000; 536/022.100; 536/023.100;
               536/023.700; 536/024.330; 536/025.320
IC      [6]
        ICM: C07H021-04
        ICS: C07H021-02; C12P019-34; C12Q001-68
EXF     536/22.1; 536/23.1; 536/23.7; 536/24.33; 536/25.32; 435/320.1; 435/6;
        435/91.2; 530/350
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L19  ANSWER 3 OF 171  USPATFULL
AN      1998:161998  USPATFULL
TI      Use of C5-specific antibodies for reducing immune and
        hemostatic dysfunctions during extracorporeal circulation
IN      Rollins, Scott, Monroe, CT, United States
        Smith, Brian R., Madison, CT, United States
        Squinto, Stephen P., Bethany, CT, United States
PA      Alexion Pharmaceuticals, Inc., New Haven, CT, United States (U.S.
        corporation)
        Yale University, New Haven, CT, United States (U.S. corporation)
PI      US 5853722                19981229
AI      US 1995-575057            19951221 (8)
RLI     Continuation of Ser. No. US 1994-217391, filed on 23 Mar 1994, now
        abandoned
DT      Utility
FS      Granted
LN.CNT 1080
INCL    INCLM: 424/145.100
        INCLS: 424/140.100; 530/387.290; 530/389.300
NCL     NCLM:  424/145.100
        NCLS:  424/140.100; 530/388.250; 530/389.300
IC      [6]
        ICM: A61K039-395
        ICS: C07K016-36
EXF     424/130.1; 424/140.1; 424/145.1; 530/387.1; 530/388.1; 530/388.25;
        530/389.1; 530/389.3
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L19  ANSWER 4 OF 171  USPATFULL
AN      1998:160100  USPATFULL
TI      ICAM-4 materials and methods
IN      Kilgannon, Patrick D., Bothell, WA, United States
        Gallatin, W. Michael, Mercer Island, WA, United States
PA      ICOS Corporation, Bothell, WA, United States (U.S. corporation)
PI      US 5852170                19981222
AI      US 1995-487595            19950607 (8)
RLI     Continuation-in-part of Ser. No. US 1994-245295, filed on 18 May 1994,
        now patented, Pat. No. US 5700658 which is a continuation-in-part of
        Ser. No. US 1993-102852, filed on 5 Aug 1993, now abandoned which is a
        continuation-in-part of Ser. No. US 1993-9266, filed on 22 Jan 1993, now
        abandoned which is a continuation-in-part of Ser. No. US 1992-894061,
        filed on 5 Jun 1992, now abandoned which is a continuation-in-part of
        Ser. No. US 1992-889724, filed on 26 May 1992, now abandoned which is a
        continuation-in-part of Ser. No. US 1992-827689, filed on 27 Jan 1992,
        now abandoned
DT      Utility
FS      Granted
LN.CNT 2389
INCL    INCLM: 530/350.000
NCL     NCLM:  530/350.000
IC      [6]
        ICM: C07K014-00
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EXF 530/350; 930/10
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L19 ANSWER 5 OF 171 USPATFULL
AN 1998:159916 USPATFULL
TI Method of enhancing proliferation or differentiation of hematopoietic stem cells using Wnt polypeptides
IN Matthews, William, Woodside, CA, United States
Austin, Timothy W., Morgan Hill, CA, United States
PA Genentech, Inc., South San Francisco, CA, United States (U.S. corporation)
PI US 5851984 19981222
AI US 1996-696566 19960816 (8)
DT Utility
FS Granted
LN.CNT 3923
INCL INCLM: 514/002.000
INCLS: 435/002.000; 424/085.100
NCL NCLM: 514/002.000
NCLS: 424/085.100; 435/002.000
IC [6]
ICM: A61K038-18
EXF 435/2; 424/85.1; 424/85.2; 514/2
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L19 ANSWER 6 OF 171 USPATFULL
AN 1998:150460 USPATFULL
TI Peripheralization of hematopoietic stem cells
IN Papayannopoulou, Thalia, Seattle, WA, United States
PA Board of Regents University of Washington, Seattle, WA, United States (U.S. corporation)
PI US 5843438 19981201
WO 9411027 19950526
AI US 1995-436339 19950713 (8)
WO 1993-US11060 19931115
19950713 PCT 371 date
19950713 PCT 102(e) date
RLI Continuation-in-part of Ser. No. US 1992-977702, filed on 13 Nov 1992, now abandoned
DT Utility
FS Granted
LN.CNT 1221
INCL INCLM: 424/130.100
INCLS: 424/133.100; 424/135.100; 424/143.100; 424/144.100; 424/152.100; 424/153.100; 424/156.100; 424/085.100; 424/085.200
NCL NCLM: 424/130.100
NCLS: 424/085.100; 424/085.200; 424/133.100; 424/135.100; 424/143.100; 424/144.100; 424/152.100; 424/153.100; 424/156.100
IC [6]
ICM: A61K039-395
ICS: A61K038-19; A61K038-21
EXF 424/130.1; 424/133.1; 424/135.1; 424/143.1; 424/144.1; 424/152.1; 424/153.1; 424/156.1; 424/85.1; 424/85.2
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L19 ANSWER 7 OF 171 USPATFULL
AN 1998:144218 USPATFULL
TI **Humanized antibodies** specific for ICAM related protein
IN Gallatin, W. Michael, Seattle, WA, United States
Vazeux, Rosemay, Seattle, WA, United States
PA ICOS Corporation, Bothell, WA, United States (U.S. corporation)
PI US 5837822 19981117
AI US 1995-487113 19950607 (8)
RLI Continuation-in-part of Ser. No. US 1993-102852, filed on 5 Aug 1993, now abandoned which is a continuation-in-part of Ser. No. US 1993-9266, filed on 22 Jan 1993, now abandoned which is a continuation-in-part of Ser. No. US 1992-894061, filed on 5 Jun 1992, now abandoned which is a continuation-in-part of Ser. No. US 1992-889724, filed on 26 May 1992, now abandoned which is a continuation-in-part of Ser. No. US 1992-827689, filed on 27 Jan 1992, now abandoned
DT Utility
FS Granted
LN.CNT 6796
INCL INCLM: 530/387.300
INCLS: 530/388.100; 530/388.220
NCL NCLM: 530/387.300
NCLS: 530/388.100; 530/388.220
IC [6]
ICM: C12P021-08
EXF 530/387.1; 530/387.3; 530/388.1; 530/388.22; 530/867
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L19 ANSWER 8 OF 171 USPATFULL
AN 1998:143928 USPATFULL
TI PYK2 related polynucleotide products
IN Schlessinger, Joseph, New York, NY, United States
Lev, Sima, San Carlos, CA, United States
PA Sugen, Inc., Redwood City, CA, United States (U.S. corporation)
New York University, New York, NY, United States (U.S. corporation)
PI US 5837524 19981117
AI US 1994-357642 19941215 (8)
DT Utility
FS Granted
LN.CNT 3585
INCL INCLM: 435/252.300
INCLS: 435/320.100; 435/091.400; 536/023.100; 536/025.400
NCL NCLM: 435/252.300
NCLS: 435/091.400; 435/320.100; 536/023.100; 536/025.400
IC [6]
ICM: C12N015-64
ICS: C12N001-21; C07H021-04
EXF 536/23.1; 536/25.4; 435/252.3; 435/320.1; 435/91.4
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L19 ANSWER 9 OF 171 USPATFULL
AN 1998:143897 USPATFULL
TI Human galectins
IN Hillman, Jennifer L., San Jose, CA, United States
Goli, Surya K., Sunnyvale, CA, United States
Bandman, Olga, Mountain View, CA, United States
Hawkins, Phillip R., Mountain View, CA, United States
Petithory, Joanne R., Fremont, CA, United States
PA Incyte Pharmaceuticals, Inc., Palo Alto, CA, United States (U.S. corporation)
PI US 5837493 19981117
AI US 1997-788584 19970123 (8)
DT Utility
FS Granted
LN.CNT 2242
INCL INCLM: 435/069.100
INCLS: 439/172.300; 439/320.100; 439/252.300; 439/325.000; 439/348.000; 439/371.000; 536/023.100; 536/023.500
NCL NCLM: 435/069.100
NCLS: 435/252.300; 435/320.100; 435/325.000; 435/348.000; 435/371.000; 536/023.100; 536/023.500
IC [6]
ICM: C12N015-00
ICS: C12N001-20; C12P021-02; C07H021-04
EXF 435/320.1; 435/69.1; 435/172.3; 435/252.3; 435/325; 435/348; 435/371;

536/23.1; 536/23.5
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L19 ANSWER 10 OF 171 USPATFULL
AN 1998:128243 USPATFULL
TI Anti-transforming growth factor-.beta. gene therapy
IN Border, Wayne A., Salt Lake City, UT, United States
PA The University of Utah, Salt Lake City, UT, United States (U.S. corporation)
PI US 5824655 19981020
AI US 1995-389887 19950215 (8)
DT Utility
FS Granted
LN.CNT 1527
INCL INCLM: 514/044.000
INCLS: 424/093.700; 424/093.210; 514/002.000; 800/002.000; 435/352.000; 435/353.000; 435/354.000; 435/366.000; 435/320.100; 435/172.300
NCL NCLM: 514/044.000
NCLS: 424/093.210; 424/093.700; 435/320.100; 435/352.000; 435/353.000; 435/354.000; 435/366.000; 514/002.000
IC [6]
ICM: A01N043-04
EXF 424/93.7; 424/93.21; 935/54; 514/2; 514/44; 800/2; 435/240.1; 435/240.2; 435/172.3; 435/320.1; 435/91.1; 435/93.1; 435/352; 435/353; 435/354; 435/366
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L19 ANSWER 11 OF 171 USPATFULL
AN 1998:127907 USPATFULL
TI Peripheralization of hematopoietic stem cells
IN Papayannopoulou, Thalia, 702 35th Ave., Seattle, WA, United States 98122
PI US 5824304 19981020
AI US 1995-463298 19950605 (8)
RLI Division of Ser. No. US 1993-436339, filed on 15 Nov 1993 which is a continuation-in-part of Ser. No. US 1992-977702, filed on 13 Nov 1992, now abandoned
DT Utility
FS Granted
LN.CNT 1214
INCL INCLM: 424/132.100
INCLS: 424/140.100; 424/144.100; 424/156.100; 424/085.100; 530/388.850; 530/389.600
NCL NCLM: 424/130.100
NCLS: 424/085.100; 424/140.100; 424/144.100; 424/156.100; 530/388.850; 530/389.600
IC [6]
ICM: A61K039-395
ICS: A61K038-19
EXF 424/85.1; 424/130.1; 424/144.1; 424/156.1; 424/140.1; 530/388.85; 530/389
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L19 ANSWER 12 OF 171 USPATFULL
AN 1998:124655 USPATFULL
TI Method for making heteromultimeric polypeptides
IN Carter, Paul J., San Francisco, CA, United States
Presta, Leonard G., San Francisco, CA, United States
Ridgway, John B., San Francisco, CA, United States
PA Genetech, Inc., South San Francisco, CA, United States (U.S. corporation)
PI US 5821333 19981013
AI US 1995-434869 19950503 (8)
RLI Division of Ser. No. US 1995-399106, filed on 1 Mar 1995
DT Utility
FS Granted
LN.CNT 2573
INCL INCLM: 530/350.000
INCLS: 530/300.000; 530/387.100; 530/387.300; 435/172.100; 435/172.300; 435/069.100; 435/069.700; 435/070.100; 435/071.100
NCL NCLM: 530/350.000
NCLS: 435/069.100; 435/069.700; 435/070.100; 435/071.100; 530/300.000; 530/387.100; 530/387.300
IC [6]
ICM: C07K001-00
EXF 435/172.1; 435/172.3; 435/69.1; 435/69.7; 435/70.1; 435/71.1; 530/300; 530/350; 530/387.1; 530/387.3
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L19 ANSWER 13 OF 171 USPATFULL
AN 1998:115830 USPATFULL
TI ICAM-related protein variants
IN Gallatin, W. Michael, Seattle, WA, United States
Vazeux, Rosemay, Seattle, WA, United States
PA ICOS Corporation, Bothell, WA, United States (U.S. corporation)
PI US 5811517 19980922
AI US 1995-483389 19950607 (8)
RLI Division of Ser. No. US 1994-286754, filed on 5 Aug 1994, now abandoned which is a continuation-in-part of Ser. No. US 1993-102852, filed on 5 Aug 1993, now abandoned which is a continuation-in-part of Ser. No. US 1993-9266, filed on 2 Dec 1993, now abandoned which is a continuation-in-part of Ser. No. US 1992-894061, filed on 5 Jun 1992, now abandoned which is a continuation-in-part of Ser. No. US 1992-889724, filed on 26 May 1992, now abandoned which is a continuation-in-part of Ser. No. US 1992-827689, filed on 27 Jan 1992, now abandoned
DT Utility
FS Granted
LN.CNT 5991
INCL INCLM: 530/350.000
INCLS: 536/023.400; 536/023.100; 435/069.100; 435/069.700; 435/320.100; 435/325.000; 435/252.300
NCL NCLM: 530/350.000
NCLS: 435/069.100; 435/069.700; 435/252.300; 435/320.100; 435/325.000; 536/023.100; 536/023.400
IC [6]
ICM: C07K019-00
ICS: C12N015-62
EXF 536/23.5; 536/23.1; 530/350; 530/395; 435/69.1; 435/69.7; 435/252.3; 435/320.1; 435/325
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L19 ANSWER 14 OF 171 USPATFULL
AN 1998:111793 USPATFULL
TI Method for making heteromultimeric polypeptides
IN Carter, Paul J., San Francisco, CA, United States
Presta, Leonard G., San Francisco, CA, United States
Ridgway, John B., San Francisco, CA, United States
PA Genentech, Inc., South San Francisco, CA, United States (U.S. corporation)
PI US 5807706 19980915
AI US 1995-433105 19950503 (8)
RLI Division of Ser. No. US 1995-399106, filed on 1 Mar 1995
DT Utility
FS Granted
LN.CNT 2576
INCL INCLM: 435/069.100
INCLS: 435/172.100; 435/172.300; 435/069.700; 435/070.100; 435/071.100; 530/300.000; 530/350.000; 530/387.100; 530/387.300
NCL NCLM: 435/069.100

NCLS: 435/069.700; 435/070.100; 435/071.100; 530/300.000; 530/350.000; 530/387.100; 530/387.300
IC [6]
ICM: C12P021-06
EXF 435/172.1; 435/172.3; 435/69.1; 435/69.7; 435/70.1; 435/71.1; 530/300; 530/350; 530/387.1; 530/387.3
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L19 ANSWER 15 OF 171 USPATFULL
AN 1998:101516 USPATFULL
TI Nucleic acids encoding protocadherin
IN Suzuki, Shintaro, Torrance, CA, United States
PA Doheny Eye Institute, Los Angeles, CA, United States (U.S. corporation)
PI US 5798224 19980825
AI US 1994-268161 19940627 (8)
RLI Continuation-in-part of Ser. No. US 1992-998003, filed on 29 Dec 1992, now patented, Pat. No. US 5643781
DT Utility
FS Granted
LN.CNT 3432
INCL INCLM: 435/069.100
INCLS: 536/023.500; 435/252.300; 435/254.110; 435/320.100; 435/325.000
NCL NCLM: 435/069.100
NCLS: 435/252.300; 435/254.110; 435/320.100; 435/325.000; 536/023.500
IC [6]
ICM: C12N015-09
EXF 435/69.1; 435/240.1; 435/252.3; 435/254.11; 435/320.1; 435/240.2; 435/325; 536/23.1; 536/23.5
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L19 ANSWER 16 OF 171 USPATFULL
AN 1998:95515 USPATFULL
TI Fibrin-binding peptide fragments of fibronectin
IN Gold, Leslie I., New York, NY, United States
Rostagno, Agueda A., Elmhurst, NY, United States
Baron, Martin, Oxford, United Kingdom
Campbell, Iain D., Oxford, United Kingdom
Williams, Michael J., Oxford, United Kingdom
PA New York University, New York, NY, United States (U.S. corporation)
Isis Innovation Ltd., Oxford, England (non-U.S. corporation)
PI US 5792742 19980811
AI US 1994-283857 19940801 (8)
RLI Continuation-in-part of Ser. No. US 1991-714134, filed on 14 Jun 1991, now abandoned
DT Utility
FS Granted
LN.CNT 4177
INCL INCLM: 514/002.000
INCLS: 424/009.100; 514/008.000; 530/350.000; 530/402.000; 435/069.600
NCL NCLM: 514/002.000
NCLS: 424/009.100; 435/069.600; 514/008.000; 530/350.000; 530/402.000
IC [6]
ICM: C07K014-78
ICS: A61K038-39
EXF 530/350; 530/380; 530/402; 530/387.1; 530/388.25; 530/389.3; 424/94.3; 424/9.1; 435/69.6; 435/188; 514/2; 514/8
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L19 ANSWER 17 OF 171 USPATFULL
AN 1998:79316 USPATFULL
TI **Humanized antibodies** reactive with GPIIB/IIIA
IN Co, Man Sung, Cupertino, CA, United States
Tso, J. Yun, Menlo Park, CA, United States
PA Protein Design Labs, Inc., Mountain View, CA, United States (U.S. corporation)
PI US 5777085 19980707
AI US 1995-458516 19950517 (8)
RLI Continuation of Ser. No. US 1993-59159, filed on 3 May 1993, now abandoned which is a continuation-in-part of Ser. No. US 1992-944159, filed on 11 Sep 1992, now abandoned which is a continuation-in-part of Ser. No. US 1992-895952, filed on 9 Jun 1992, now abandoned which is a continuation-in-part of Ser. No. US 1991-812111, filed on 20 Dec 1991, now abandoned
DT Utility
FS Granted
LN.CNT 1486
INCL INCLM: 530/388.230
INCLS: 530/287.300; 530/388.700; 435/069.100; 435/172.300; 435/320.100; 435/326.000; 435/328.000; 435/334.000; 435/343.000; 536/023.530
NCL NCLM: 530/388.230
NCLS: 435/069.100; 435/320.100; 435/326.000; 435/328.000; 435/334.000; 435/343.000; 530/387.300; 530/388.700; 536/023.530
IC [6]
ICM: C07K016-18
ICS: C07K016-28; C12P005-10; C07H021-04
EXF 424/130.1; 424/133.1; 424/134.1; 424/141.1; 424/143.1; 424/145.1; 424/152.1; 424/172.1; 435/70.21; 435/171.2; 435/69.1; 435/172.3; 435/320.1; 435/310.1; 536/23.5; 536/23.53; 530/387.1; 530/387.3; 530/388.1; 530/388.7; 530/388.23
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L19 ANSWER 18 OF 171 USPATFULL
AN 1998:75442 USPATFULL
TI Anti-ICAM-4 **antibodies** and hybridomas
IN Kilgannon, Patrick D., Bothell, WA, United States
Gallatin, W. Michael, Mercer Island, WA, United States
PA ICOS Corporation, Bothell, WA, United States (U.S. corporation)
PI US 5773293 19980630
AI US 1995-485604 19950607 (8)
RLI Continuation-in-part of Ser. No. US 1994-245295, filed on 18 May 1994, now patented, Pat. No. US 5700658 which is a continuation-in-part of Ser. No. US 1993-102852, filed on 5 Aug 1993, now abandoned which is a continuation-in-part of Ser. No. US 1993-9266, filed on 22 Jan 1993, now abandoned which is a continuation-in-part of Ser. No. US 1992-894061, filed on 5 Jun 1992, now abandoned which is a continuation-in-part of Ser. No. US 1992-889724, filed on 26 May 1992, now abandoned which is a continuation-in-part of Ser. No. US 1992-827689, filed on 27 Jan 1992, now abandoned
DT Utility
FS Granted
LN.CNT 1896
INCL INCLM: 435/334.000
INCLS: 435/070.210; 530/388.100; 530/388.220
NCL NCLM: 435/334.000
NCLS: 435/070.210; 530/388.100; 530/388.220
IC [6]
ICM: C12N005-12
ICS: C07K016-00; C12P021-08
EXF 530/387.1; 530/387.9; 530/388.1; 530/388.15; 530/389.1; 530/388.22; 435/70.1; 435/70.2; 435/70.21; 435/240.26; 435/240.27; 435/334
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L19 ANSWER 19 OF 171 USPATFULL
AN 1998:75369 USPATFULL
TI Method to identify compounds which modulate ICAM-related protein interactions
IN Gallatin, W. Michael, Seattle, WA, United States
Vazeux, Rosemay, Seattle, WA, United States
PA ICOS Corporation, Bothell, WA, United States (U.S. corporation)
PI US 5773218 19980630

AI US 1995-482882 19950607 (8)
RLI Division of Ser. No. US 1994-286754, filed on 5 Aug 1994 which is a continuation-in-part of Ser. No. US 1993-102852, filed on 5 Aug 1993, now abandoned which is a continuation-in-part of Ser. No. US 1993-9266, filed on 22 Jan 1993, now abandoned And Ser. No. US 1992-894061, filed on 5 Jun 1992, now abandoned which is a continuation-in-part of Ser. No. US 1992-889724, filed on 26 May 1992, now abandoned which is a continuation-in-part of Ser. No. US 1992-827689, filed on 27 Jan 1992, now abandoned
DT Utility
FS Granted
LN.CNT 5498
INCL INCLM: 435/006.000
NCL NCLM: 435/006.000
IC [6]
ICM: C12Q001-68
EXF 435/6; 435/7.2; 435/69.1; 536/23.5
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L19 ANSWER 20 OF 171 USPATFULL
AN 1998:72709 USPATFULL
TI ICAM-related protein fragments
IN Gallatin, W. Michael, Seattle, WA, United States
Vazeux, Rosemay, Seattle, WA, United States
PA ICOS Corporation, Bothell, WA, United States (U.S. corporation)
PI US 5770686 19980623
AI US 1995-474368 19950607 (8)
RLI Division of Ser. No. US 1995-425870, filed on 20 Apr 1995, now abandoned which is a continuation of Ser. No. US 1993-102852, filed on 5 Aug 1993, now abandoned which is a continuation-in-part of Ser. No. US 1993-9266, filed on 22 Jan 1993, now abandoned which is a continuation-in-part of Ser. No. US 1992-894061, filed on 5 Jun 1992, now abandoned which is a continuation-in-part of Ser. No. US 1992-889724, filed on 26 May 1992, now abandoned which is a continuation-in-part of Ser. No. US 1992-827689, filed on 27 Jan 1992, now abandoned
PRAI WO 1993-US787 19930126
DT Utility
FS Granted
LN.CNT 3927
INCL INCLM: 530/300.000
INCLS: 530/317.000; 530/330.000; 530/350.000; 530/395.000
NCL NCLM: 530/300.000
NCLS: 530/317.000; 530/330.000; 530/350.000; 530/395.000
IC [6]
ICM: C07K014-705
EXF 530/300; 530/350; 530/395; 530/330; 530/317
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L19 ANSWER 21 OF 171 USPATFULL
AN 1998:65006 USPATFULL
TI Gene encoding a human reduced folate carrier (RFC)
IN Moscow, Jeffrey A., Silver Spring, MD, United States
Cowan, Kenneth H., Potama, MD, United States
Dixon, Kathy, Olney, MD, United States
He, Rui, Germantown, MD, United States
PA The United States of America as represented by the Department of Health & Human Services, Washington, DC, United States (U.S. government)
PI US 5763216 19980609
AI US 1995-483094 19950607 (8)
DT Utility
FS Granted
LN.CNT 1468
INCL INCLM: 435/069.100
INCLS: 435/320.100; 536/023.500
NCL NCLM: 435/069.100
NCLS: 435/006.000; 435/320.100; 536/023.500
IC [6]
ICM: C12Q001-68
ICS: C12P021-00; C12N015-09; C12N015-12
EXF 536/23.5; 536/24.1; 435/320.1; 435/69.1; 435/252.3; 435/254.11; 435/325; 435/6
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L19 ANSWER 22 OF 171 USPATFULL
AN 1998:54748 USPATFULL
TI Neuron-specific ICAM-4 promoter
IN Kilgannon, Patrick D., Bothell, WA, United States
Gallatin, W. Michael, Mercer Island, WA, United States
PA ICOS Corporation, Bothell, WA, United States (U.S. corporation)
PI US 5753502 19980519
AI US 1996-656984 19960606 (8)
RLI Continuation-in-part of Ser. No. US 1995-481130, filed on 7 Jun 1995, now patented, Pat. No. US 5702917 which is a continuation-in-part of Ser. No. US 1994-245295, filed on 18 May 1994, now patented, Pat. No. US 5700658 which is a continuation-in-part of Ser. No. US 1993-102852, filed on 5 Aug 1993, now abandoned
DT Utility
FS Granted
LN.CNT 2173
INCL INCLM: 435/320.100
INCLS: 435/325.000; 536/024.100
NCL NCLM: 435/320.100
NCLS: 435/325.000; 536/024.100
IC [6]
ICM: C12N014-85
EXF 536/24.1; 435/320.1; 435/325; 435/252.3
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L19 ANSWER 23 OF 171 USPATFULL
AN 1998:48195 USPATFULL
TI Method and device for diagnosing and distinguishing chest pain in early onset thereof
IN Jackowski, George, Inglewood, Canada
PA Spectral Diagnostics Inc., Toronto, Canada (non-U.S. corporation)
PI US 5747274 19980505
AI US 1996-697690 19960905 (8)
RLI Continuation of Ser. No. US 1995-420298, filed on 11 Apr 1995, now patented, Pat. No. US 5604105 which is a continuation-in-part of Ser. No. US 1993-26453, filed on 3 Mar 1993, now abandoned which is a continuation-in-part of Ser. No. US 1991-695381, filed on 3 May 1991, now patented, Pat. No. US 5290678, issued on 1 Mar 1994
PRAI CA 1990-2027434 19901012
DT Utility
FS Granted
LN.CNT 2438
INCL INCLM: 435/007.940
INCLS: 422/056.000; 422/058.000; 422/060.000; 422/061.000; 435/007.930; 435/007.940; 435/970.000; 435/973.000; 435/975.000; 436/514.000; 436/528.000; 436/530.000; 436/531.000; 436/161.000; 436/164.000; 436/807.000; 436/808.000; 436/810.000; 436/811.000
NCL NCLM: 435/007.940
NCLS: 422/056.000; 422/058.000; 422/060.000; 422/061.000; 435/007.930; 435/970.000; 435/973.000; 435/975.000; 436/161.000; 436/164.000; 436/514.000; 436/528.000; 436/530.000; 436/531.000; 436/807.000; 436/808.000; 436/810.000; 436/811.000
IC [6]
ICM: G01N033-573
ICS: G01N033-558
EXF 422/55; 422/56; 422/58; 422/60; 422/61; 435/7.9; 435/7.92; 435/7.93; 435/7.94; 435/7.4; 435/969; 435/970; 435/973; 435/975; 436/514; 436/528;

436/530; 436/531; 436/161; 436/164; 436/807; 436/808; 436/810; 436/811
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L19 ANSWER 24 OF 171 USPATFULL
AN 1998:47965 USPATFULL
TI Polypeptides with increased half-life for use in treating disorders involving the LFA-1 receptor
IN Presta, Leonard G., San Francsico, CA, United States
Snedecor, Bradley R., Portola Valley, CA, United States
PA Genentech, Inc., South San Francisco, CA, United States (U.S. corporation)
PI US 5747035 19980505
AI US 1995-422091 19950414 (8)
DT Utility
FS Granted
LN.CNT 3305
INCL INCLM: 424/144.100
INCLS: 424/130.100; 424/133.100; 424/135.100; 424/141.100; 424/143.100; 424/153.100; 424/154.100; 424/173.100; 514/002.000; 514/008.000; 514/885.000; 530/387.100
NCL NCLM: 424/144.100
NCLS: 424/130.100; 424/133.100; 424/135.100; 424/141.100; 424/143.100; 424/153.100; 424/154.100; 424/173.100; 514/002.000; 514/008.000; 514/885.000; 530/387.100
IC [6]
ICM: A61K039-395
ICS: A61K038-02; A61K038-17
EXF 424/130.1; 424/133.1; 424/135.1; 424/144.1; 424/173.1; 424/141.1; 424/143.1; 424/153.1; 424/154.1; 514/2; 514/8; 514/835; 530/387.1
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L19 ANSWER 25 OF 171 USPATFULL
AN 1998:45097 USPATFULL
TI Method and device for diagnosing and distinguishing chest pain in early onset thereof
IN Jackowski, George, Inglewood, Canada
PA Spectral Diagnostics Inc., Toronto, Canada (non-U.S. corporation)
PI US 5744358 19980428
AI US 1996-707594 19960905 (8)
RLI Continuation of Ser. No. US 1995-420298, filed on 11 Apr 1995, now patented, Pat. No. US 5604105 which is a continuation-in-part of Ser. No. US 1993-26453, filed on 3 Mar 1993, now abandoned which is a continuation-in-part of Ser. No. US 1991-695381, filed on 3 May 1991, now patented, Pat. No. US 5290678, issued on 1 Mar 1994
PRAI CA 1990-2027434 19901012
DT Utility
FS Granted
LN.CNT 2396
INCL INCLM: 435/007.400
INCLS: 422/056.000; 422/058.000; 422/060.000; 422/061.000; 435/007.940; 435/970.000; 435/973.000; 435/975.000; 436/514.000; 436/528.000; 436/530.000; 436/531.000; 436/161.000; 436/164.000; 436/807.000; 436/808.000; 436/810.000; 436/811.000
NCL NCLM: 435/007.400
NCLS: 422/056.000; 422/058.000; 422/060.000; 422/061.000; 435/007.940; 435/970.000; 435/973.000; 435/975.000; 436/161.000; 436/164.000; 436/514.000; 436/528.000; 436/530.000; 436/531.000; 436/807.000; 436/808.000; 436/810.000; 436/811.000
IC [6]
ICM: G01N033-573
ICS: G01N033-558
EXF 422/55; 422/56; 422/58; 422/60; 422/61; 435/7.9; 435/7.92; 435/7.94; 435/7.4; 435/969; 435/970; 435/973; 435/975; 436/514; 436/528; 436/530; 436/531; 436/161; 436/164; 436/807; 436/808; 436/810; 436/811
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L19 ANSWER 26 OF 171 USPATFULL
AN 1998:42455 USPATFULL
TI Protein binding fragments of gravin
IN Scott, John D., Portland, OR, United States
Nauert, J. Brian, Portland, OR, United States
Klauck, Theresa M., Portland, OR, United States
PA Oregon Health Sciences University, Portland, OR, United States (U.S. corporation)
PI US 5741890 19980421
AI US 1996-769309 19961219 (8)
DT Utility
FS Granted
LN.CNT 1567
INCL INCLM: 530/300.000
INCLS: 530/324.000; 530/350.000; 435/691.000
NCL NCLM: 530/300.000
NCLS: 435/069.100; 530/324.000; 530/350.000
IC [6]
ICM: C07K014-00
EXF 530/350; 530/300; 530/324; 435/69.1
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L19 ANSWER 27 OF 171 USPATFULL
AN 1998:39693 USPATFULL
TI gC1q receptor, HIV-1 gp120 region binding thereto, and related peptides and targeting **antibodies**
IN Fung, Michael S.C., Houston, TX, United States
Sun, Bill N.C., Bellaire, TX, United States
Sun, Cecily R.Y., Bellaire, TX, United States
Kim, Young Woo, Plainsboro, NJ, United States
Yu, Liming, Houston, TX, United States
PA Tanox Biosystems, Inc., Houston, TX, United States (U.S. corporation)
PI US 5739306 19980414
AI US 1996-711175 19960909 (8)
RLI Division of Ser. No. US 1995-410360, filed on 24 Mar 1995
DT Utility
FS Granted
LN.CNT 1155
INCL INCLM: 536/023.500
INCLS: 424/185.100; 424/188.100; 424/208.100; 514/013.000; 514/014.000; 530/326.000; 530/327.000; 530/395.000; 536/023.100; 536/023.720
NCL NCLM: 536/023.500
NCLS: 424/185.100; 424/188.100; 424/208.100; 530/326.000; 530/327.000; 530/395.000; 536/023.100; 536/023.720
IC [6]
ICM: C07H021-04
EXF 424/185.1; 424/188.1; 424/208.1; 514/13; 514/14; 530/326; 530/327; 530/395; 536/23.1; 536/23.5; 536/23.72
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L19 ANSWER 28 OF 171 USPATFULL
AN 1998:39666 USPATFULL
TI Altered polypeptides with increased half-life
IN Presta, Leonard G., San Francisco, CA, United States
Snedecor, Bradley R., Portola Valley, CA, United States
PA Genentech Inc., San Francisco, CA, United States (U.S. corporation)
PI US 5739277 19980414
AI US 1995-422101 19950414 (8)
DT Utility
FS Granted
LN.CNT 3251
INCL INCLM: 530/326.000
INCLS: 530/300.000; 530/350.000; 530/387.100
NCL NCLM: 530/326.000

NCLS: 530/300.000; 530/350.000; 530/387.100
IC [6]
ICM: C07K007-08
ICS: C07K014-47; C07K016-00; C07K016-46
EXF 530/327; 530/328; 530/387.1; 530/300; 530/350; 530/326
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L19 ANSWER 29 OF 171 USPATFULL
AN 1998:36551 USPATFULL
TI Secreted Mac-2-binding glycoprotein
IN Koths, Kirston E., El Cerrito, CA, United States
Halenbeck, Robert F., San Rafael, CA, United States
Taylor, Eric W., Berkeley, CA, United States
Wang, Alice M., Lafayette, CA, United States
Casipit, Clayton L., Hayward, CA, United States
PA Chiron Corporation, Emeryville, CA, United States (U.S. corporation)
PI US 5736340 19980407
AI US 1995-473791 19950607 (8)
RLI Division of Ser. No. US 1994-316714, filed on 29 Sep 1994 which is a continuation of Ser. No. US 1992-961404, filed on 15 Oct 1992, now abandoned which is a continuation-in-part of Ser. No. US 1991-777121, filed on 16 Oct 1991, now abandoned
DT Utility
FS Granted
LN.CNT 1706
INCL INCLM: 435/007.100
INCLS: 435/007.200; 435/007.230; 436/063.000; 436/064.000; 436/813.000
NCL NCLM: 435/007.100
NCLS: 435/007.200; 435/007.230; 436/063.000; 436/064.000; 436/813.000
IC [6]
ICM: G01N033-53
EXF 435/7.23; 435/7.2; 435/7.1; 436/63; 436/64; 436/813
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L19 ANSWER 30 OF 171 USPATFULL
AN 1998:30879 USPATFULL
TI Method for making heteromultimeric polypeptides
IN Carter, Paul J., San Francisco, CA, United States
Presta, Leonard G., San Francisco, CA, United States
Ridgway, John B., San Francisco, CA, United States
PA Genentech, Inc., South San Francisco, CA, United States (U.S. corporation)
PI US 5731168 19980324
AI US 1995-399106 19950301 (8)
DT Utility
FS Granted
LN.CNT 2657
INCL INCLM: 435/069.100
INCLS: 435/172.100; 435/172.300; 435/070.100; 435/071.100; 435/069.700; 530/300.000; 530/350.000; 530/387.100; 530/387.300; 536/023.100; 536/023.400; 536/023.500; 536/023.530
NCL NCLM: 435/069.100
NCLS: 435/069.700; 435/070.100; 435/071.100; 530/300.000; 530/350.000; 530/387.100; 530/387.300; 536/023.100; 536/023.400; 536/023.500; 536/023.530
IC [6]
ICM: C12D021-06
EXF 435/172.1; 435/172.3; 435/69.1; 435/70.1; 435/71.1; 435/69.7; 530/300; 530/350; 530/387.1; 530/387.3; 536/23.1; 536/23.4; 536/23.5; 536/23.53
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

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Executing the logoff script...

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COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	177.17	177.38

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	-0.65	-0.65

STN INTERNATIONAL LOGOFF AT 14:17:20 ON 24 APR 2003